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THE EFFECT OF HETERO-SPECIFIC PREGNANCY ON THE HÆMOGLOBIN AND RED CELL LEVELS IN THE NEWBORN INFANT.

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DURING an earlier study of maternal and infantile blood factor relationships (Bryce, Jakobowicz and McArthur, 1946) it was noted that A B O incompatibility between the blood of the mother and that of the child appeared to be associated with a higher incidence in the infant of anæmia of the so-called "late" type than did Rh incompatibility. The term "late" anæmia is used to indicate a relatively mild type, usually developing about the second to sixth week after birth. The number of cases included in that study was, however, too small for effective comparison, and as the point appeared to us to be of some importance, a larger series has been examined with special reference to the development of anæmia in the infant during the first few weeks after birth.

The data presented are based on estimation of the hæmoglobin level and red cell counts in the blood of 409 infants whom we classified in the following groups: Group I: 156 in whom both the A B O and Rh factors were the same as or compatible (as defined in our previous paper, 1946) with those of the mother; Group II: 154 in whom the A B O factors were incompatible with those of the mother and the Rh factors were the same or compatible; Group III: 66 with A B O factors the same as or compatible with those of the mother and incompatible Rh factors; Group IV: 33 having both A B O and Rh factors incompatible with those of the mother.

The hæmoglobin level of all infants was estimated within twenty-four hours of birth. Similar estimations were made on 400 of these babies on the third day after birth and on 372 during the sixth week of post-natal life. The blood of 94 babies was also examined periodically for three months or more after birth.

The hæmoglobin estimations were made with a Sahli instrument standardized by comparison with photo-electric measurements by Mr. H. F. Holden, and oxygen capacity estimations carried out by the Van Slyke method by Miss Beryl Splatt; to both of these workers we are greatly indebted for their invaluable assistance. A correction factor was applied to give readings in which 100% on our Sahli scale was equivalent to 14 grammes of hæmoglobin per 100 millilitres of blood. The pipettes and tubes were tested for accuracy. The blood samples were obtained from heel pricks after the promotion of free circulation by warming and gentle friction.

The accuracy of the Sahli (acid hæmatin) method has been questioned; but in our hands, when due consideration has been given to standardization of the instrument, to calibration of pipettes and tubes, and to avoidance of errors in the technique of sampling, results have been sufficiently reproducible to give us confidence that the inherent error of the method does not, in a series of the size reported in this paper, significantly alter the observed differences between the mean values obtained in each of our four groups.

[1] The salary (part-time) of one of us (L.K.) and clerical expenses were defrayed by a grant from the National Health and Medical Research Council.

[2] A preliminary report on this work was read at a scientific conference of the National Red Cross Blood Transfusion Service on June 12, 1947.

TABLE Ia.

Hæmoglobin Levels of Infants on the First and Third Days and Six Weeks after Birth.

Relationship of Maternal and Infantile Blood Factors.	First Day.				Third Day.				Sixth Week.			
	Number of Cases.	Mean Hæmoglobin Value.[1]	Standard Error of Mean.	Standard Deviation.	Number of Cases.	Mean Hæmoglobin Value.[1]	Standard Error of Mean.	Standard Deviation.	Number of Cases.	Mean Hæmoglobin Value.[1]	Standard Error of Mean.	Standard Deviation.
Group I. A B O compatible, Rh compatible.	156	151·3%	1·1	12	147	148·7%	1·2	11	132	95·2%	1·2	15
Group II. A B O incompatible, Rh compatible.	154	145·9%	1·2	16	156	140·1%	1·1	16	156	85·4%	1·1	13
Group III. A B O compatible, Rh incompatible.	66	143·1%	1·8	16	66	137·6%	1·8	17	56	87·8%	1·9	15
Group IV. A B O incompatible, Rh incompatible.	33	140·0%	2·5	16	31	139·0%	2·6	13	28	84·1%	2·7	14

[1] The hæmoglobin percentage is in terms of a standard in which 100% is equivalent to 14 grammes per 100 millilitres.

Only babies judged to have been born at full term were included. Apart from this degree of selection (which aimed at exclusion of cases of anæmia due to prematurity as such) and the omission of cases in Groups III and IV in which Rh antibodies were demonstrable in the maternal serum, the cases were consecutive in their respective groups. The cases in Groups III and IV were those occurring during the period necessary to obtain approximately equal numbers in Groups I and II.

With very few exceptions the mothers had attended the ante-natal department and had therefore received iron therapy if indicated.

The blood groups of the mothers and babies, the agglutinin content of the maternal serum, with special reference to rise in titre during the puerperium, and the secretor or non-secretor status of some of the babies were determined, as it was thought that these factors might play a part in the production of anæmia.

The incidence of clinically detectable jaundice or splenomegaly, or of both, in 336 babies (not all included in the above series) was recorded. It was thought possible that the presence of these features might bear some significant relationship to blood-group incompatibility between mother and child.

TABLE Ib.

Hæmoglobin Estimations.

(100% = 14 grammes per 100 millilitres.)

Range of Hæmoglobin Values.	Number of Cases.					Group Range.
	Group I.	Group II.	Group III.	Group IV.	Total.	
First day:						
161% to 170% ..	30	28	9	2	69	
151% to 160% ..	65	40	15	6	126	
141% to 150% ..	40	36	16	11	103	Group I: 170% to 106%.
131% to 140% ..	14	26	13	8	61	Group II: 165% to 91%.
121% to 130% ..	2	14	7	2	25	Group III: 165% to 101%.
111% to 120% ..	3	6	2	1	12	Group IV: 161% to 91%.
101% to 110% ..	2	2	4	2	10	
91% to 100% ..	0	2	0	1	3	
Total	156	154	66	33	409	
Third day:						
161% to 170% ..	13	14	3	0	30	
151% to 160% ..	66	33	15	6	120	
141% to 150% ..	40	35	13	10	98	Group I: 165% to 101%.
131% to 140% ..	15	30	13	8	66	Group II: 165% to 87%.
121% to 130% ..	10	28	12	5	55	Group III: 165% to 100%.
111% to 120% ..	2	7	5	0	14	Group IV: 159% to 110%.
101% to 110% ..	1	8	4	2	15	
91% to 100% ..	0	0	1	0	1	
81% to 90% ..	0	1	0	0	1	
Total	147	156	66	31	400	
Sixth week:						
121% to 130% ..	7	1	3	1	12	
111% to 120% ..	17	1	1	0	19	
101% to 110% ..	21	17	8	2	48	Group I: 123% to 62%.
91% to 100% ..	36	35	8	4	83	Group II: 128% to 57%.
81% to 90% ..	25	45	14	10	94	Group III: 123% to 62%.
71% to 80% ..	23	40	18	8	89	Group IV: 123% to 60%.
61% to 70% ..	3	14	4	1	22	
51% to 60% ..	0	3	0	2	5	
Total	132	156	56	28	372	

TABLE Ic.
Statistical Analysis of Hæmoglobin Levels.

Time of Examination.	A B O Relationship (Mother and Infant).	Rh Relationship (Mother and Infant).			
		Rh Compatible.	Rh Not Compatible.	Difference.	P[1]
First day.	A B O compatible	151·3	143·1	8·2	*<0·001*
	A B O not compatible	145·9	140·0	5·9	*≃0·05*
	Difference	5·4	3·1	11·3	
	P	*<0·001*	0·38		
Third day.	A B O compatible	148·7	137·6	11·1	*<0·001*
	A B O not compatible	140·1	139·0	1·1	
	Difference	8·6	−1·4	10·0	
	P	*<0·001*			
Sixth week.	A B O compatible	95·2	87·8	7·4	*0·001* to *0·01*
	A B O not compatible	85·5	84·1	1·4	
	Difference	9·7	3·7	6·0	
	P	*<0·001*	0·29		

[1] P = the probability that a difference as large or larger than that observed would occur by chance alone in groups selected from the same basic population.

Results.

The mean hæmoglobin level, the standard error of the mean, and the standard deviation in each group, on the first and third days and in the sixth week after birth, are shown in Table IA. The range of observed hæmoglobin levels is shown in Table IB, and the statistical analysis of the data is summarized in Table Ic.

Group I is the control series in relation to the other groups, in that it is comprised of newborn full-term babies, whose blood picture could not be influenced by A B O or Rh blood factor incompatibilities, and who therefore, from this aspect at least, are normal. It will be seen that the mean hæmoglobin level in these babies is 151 (± 1)%, which is equivalent to 21·14 ± 0·14 grammes per 100 millilitres with a standard deviation of 12, or, in terms of grammes, 1·68.

In a review by an American authority, Clement A. Smith (1946), the majority of quoted studies from American and European sources show hæmoglobin levels on the first day of between 17·2 and 23·2 grammes per 100 millilitres, and in a recent paper Potter and Bernstein (1948) found the mean hæmoglobin level of normal infants on the first day to be 20·7 grammes per 100 millilitres, with a range from 15·2 to 26·2 grammes. The work of Helen MacKay (1933) is generally considered to be the most authoritative as regards English infants. She found that in one series examined by her, comprising 62 healthy babies born in London, the average hæmoglobin level within twenty-four hours of birth was 145·7% on the Haldane scale, which at that time was considered to be equivalent to 20·18 grammes per 100 millilitres. If this reading was recalculated in the light of recent investigations by E. J. King *et alii* (1947) on the Haldane scale it would be equivalent to 21·56 grammes per 100 millilitres. Our own figure of 151% is equal to 21·14 grammes per 100 millilitres.

Comparison of the control series (Group I) with Groups II, III and IV (Table I) shows that in each of the latter—that is, among babies whose A B O or Rh factors or both are incompatible with these factors in their mothers—the hæmoglobin levels are lower on the first and third days and in the sixth week than those in Group I, in which there is no incompatibility. Statistical analysis indicates that the differences between the hæmoglobin levels in Group I and those in Groups II, III and IV are significant, while the smaller differences between the three last mentioned are not. Although it is not statistically significant, there is also an indication of a wider range of divergence from the mean in the three last mentioned, as shown by the standard deviations. This indication is manifest on the first and third days, but is not apparent by the sixth week. Further analysis (Table Ic) indicates that in cases in which A B O compatibility is present, there is a significant difference between the mean hæmoglobin levels of babies in Groups I and III; that is, Rh incompatibility appears to cause a reduction in the mean hæmoglobin level both on the first and third days and in the sixth week. Similarly, A B O incompatibility (without Rh incompatibility) causes a lowering of the mean hæmoglobin level at each of these times. A double incompatibility, however, appears from this analysis to cause a lowering of mean hæmoglobin level not greatly in excess of that caused by a single incompatibility and of a doubtful interpretation. Although, as was indicated above, the hæmoglobin level during the sixth week is significantly lower in groups with incompatibilities than in the control group, in all four the level in the sixth week is lower than that shortly after birth. This is in accord with the well-established fact that during the first three months of extrauterine life there is in all babies a progressive lowering of the hæmoglobin level, usually to a minimum somewhere between 70% and 80% (9·8 to 11·2 grammes per 100 millilitres).

The observation that the colour indices (Table IIA) in the sixth week all approximate to unity would seem to indicate that whatever process caused the differences observed in the original hæmoglobin levels has ceased and has been compensated for by this time, the actual levels in this sixth week being thus a reflection of those with which the infant started its extrauterine life.

Of the 94 babies whose examination was extended beyond the sixth week, the average hæmoglobin level at the twelfth week was 77% and the colour index approximately 1·0, a finding which is in accord with that of Helen MacKay and others. Statistical comparison of the figures obtained in this series is not, however, possible, as there was selection of those who appeared to need further blood examinations, either on clinical grounds or because the hæmoglobin level at six weeks was 80% or less. From this aspect the fact that the babies examined comprise only 9% of the total number in Group I, but 27%, 38% and 31% of Groups II, III and IV respectively, may be significant. The lowest hæmoglobin level found, without any obvious association with intercurrent infection or other possible extraneous cause, was 56% (7·8 grammes per 100 millilitres) with a red cell count of 3,010,000 per cubic millimetre (colour index 0·93) at the ninth week, in a baby in Group II; this baby was treated by blood transfusion. Blood transfusions were also given to three others in this group, whose hæmoglobin levels were 57%, 62% and 57% at the sixth, twelfth and thirteenth weeks respectively, with red cell counts below 3,000,000 per cubic millimetre (colour indices of 1·19, 1·18 and 1·04 respectively), and to one baby in the control group whose hæmoglobin level at the sixth week was 61% (8·5 grammes per 100 millilitres) with a red cell count of 2,900,000 per cubic millimetre (colour index 1·05).

The mean colour indices in each group at the first and third days and in the sixth week are shown in Table IIA and the calculated figures and range in Table IIB. Statistical analysis shows a significant difference between the mean colour index of Group I and the mean colour indices of Groups II, III and IV on the first and third days.

The mean red cell counts are shown in Table IIIA. The observed figures and range (Table IIIB) are essentially

TABLE IIA.
Colour Indices of Infants on First and Third Days, and Six Weeks after Birth.

Relationship of Maternal and Infantile Blood Factors.	First Day.				Third Day.				Sixth Week.			
	Number of Cases.	Mean Colour Index.	Standard Error of Mean.	Standard Deviation.	Number of Cases.	Mean Colour Index.	Standard Error of Mean.	Standard Deviation.	Number of Cases.	Mean Colour Index.	Standard Error of Mean.	Standard Deviation.
Group I. A B O compatible, Rh compatible.	156	1·31	0·01	0·14	147	1·32	0·01	0·13	55	1·08	0·01	0·11
Group II. A B O incompatible, Rh compatible.	153	1·24	0·01	0·13	156	1·23	0·01	0·12	156	1·07	0·01	0·11
Group III. A B O compatible, Rh incompatible.	63	1·21	0·02	0·15	66	1·22	0·02	0·13	56	1·08	0·01	0·11
Group IV. A B O incompatible, Rh incompatible.	31	1·12	0·02	0·11	31	1·22	0·02	0·14	28	1·04	0·02	0·11

similar in each of the four groups. These figures agree reasonably closely both with those of Sanford (quoted by Clement Smith, 1946), who found that in a series of 200 newborn infants the red cell count varies from 4,200,000 to 8,000,000 per cubic millimetre with a mean of 5,800,000 per cubic millimetre, and with those of Potter and Bernstein (1948).

In England it is generally accepted, according to Whitby and Britton (1946), that the average red cell count shortly after birth is between 6,500,000 and 7,500,000 per cubic millimetre.

By the sixth week there is no significant difference in our series between the ranges or the means of the colour indices and the corresponding red cell counts in the "compatible" and "incompatible" groups.

No correlation could be demonstrated between a puerperal rise in agglutinin titre in the maternal serum, or the secretor status of the infant, and the presence of "late" anæmia.

In this series no correlation could be shown between blood group incompatibility of mother and child and the incidence of clinically detectable jaundice and splenomegaly (Table IV).

Discussion.

Our investigations show that in babies with A B O or Rh blood factors incompatible with those of their mothers the hæmoglobin levels and colour indices are lower than those in babies of the control group, whereas the red cell counts are almost identical in each group. These findings suggest

TABLE IIB.
Colour Indices.[1]

Range of Colour Indices.	Number of Cases.					Group Range.
	Group I.	Group II.	Group III.	Group IV.	Total.	
First day:						
1·51 to 1·60 ..	11	1	1	0	13	
1·41 to 1·50 ..	27	9	4	0	40	
1·31 to 1·40 ..	51	42	10	4	107	Group I: 1·56 to 0·88.
1·21 to 1·30 ..	39	52	25	13	129	Group II: 1·52 to 0·90.
1·11 to 1·20 ..	15	27	11	8	61	Group III: 1·55 to 0·90.
1·01 to 1·10 ..	7	14	5	4	30	Group IV: 1·40 to 0·92.
0·91 to 1·00 ..	4	7	5	2	18	
0·81 to 0·90 ..	2	1	2	0	5	
Total	156	153	63	31	403	
Third day:						
1·51 to 1·60 ..	11	1	0	0	12	
1·41 to 1·50 ..	28	10	3	2	43	
1·31 to 1·40 ..	49	27	13	7	96	Group I: 1·57 to 0·94.
1·21 to 1·30 ..	37	58	28	11	134	Group II: 1·51 to 0·95.
1·11 to 1·20 ..	14	37	12	5	68	Group III: 1·46 to 0·86.
1·01 to 1·10 ..	2	16	5	4	27	Group IV: 1·46 to 0·90.
0·91 to 1·00 ..	6	7	3	1	17	
0·81 to 0·90 ..	0	0	2	1	3	
Total	147	156	66	31	400	
Sixth week:						
1·31 to 1·40 ..	2	2	3	0	7	
1·21 to 1·30 ..	3	11	5	3	22	Group I: 1·40 to 0·87.
1·11 to 1·20 ..	16	46	12	3	77	Group II: 1·40 to 0·81.
1·01 to 1·10 ..	22	51	21	11	105	Group III: 1·40 to 0·87.
0·91 to 1·00 ..	11	37	14	10	72	Group IV: 1·26 to 0·85.
0·81 to 0·90 ..	1	9	1	1	12	
Total	55	156	56	28	295	

[1]In the calculation of the colour indices, 5,000,000 red cells per cubic millimetre = 100%.

TABLE IIIA
Red Cell Counts.

Relationship of Maternal and Infantile Blood Factors.	First Day.			Third Day.			Sixth Week.		
	Mean Red Cell Count.	Standard Error of Mean.	Standard Deviation.	Mean Red Cell Count.	Standard Error of Mean.	Standard Deviation.	Mean Red Cell Count.	Standard Error of Mean.	Standard Deviation.
Group I: A B O compatible, Rh compatible	5·78	0·04	0·48	5·64	0·04	0·45	4·05	0·09	0·65
Group II: A B O incompatible, Rh compatible ..	5·82	0·05	0·62	5·76	0·05	0·58	3·98	0·05	0·63
Group III: A B O compatible, Rh incompatible ..	5·83	0·09	0·76	5·78	0·08	0·65	4·08	0·09	0·68
Group IV: A B O incompatible, Rh incompatible ..	5·72	0·11	0·63	5·59	0·09	0·50	4·04	0·12	0·65

changes akin in a mild degree to the hypochromia and microcytosis which in later life are produced by iron deficiency. It is usually assumed, however, that the action of incompatible maternal blood factor antibodies on the fœtal cells is hæmolytic. In adults, intravascular hæmolysis does not usually result in appreciable lowering of the colour index. A possible explanation which would reconcile a hæmolytic process with the trend towards hypochromia and microcytosis as found in these infants is that the iron liberated from the fœtal cells becomes distributed through both fœtal and the relatively much larger maternal circulations, and that in the latter it may be, partly at any rate, utilized for maternal erythropoiesis. Other possibilities are that the maternal antibodies may have a deleterious effect not only on the red cells, with resultant hæmolysis, but also on other cells, one result of which is to render the fœtal hæmatopoietic tissues unable to resynthesize the liberated iron, or that, as a result of persistent destructive action by the antibodies, fœtal red cells as they are developing are broken down before hæmoglobin formation is complete.

The above three interpretations postulate a hæmolytic process as an element in the production of the infantile anæmia. There is no doubt that in the severe manifestations of Rh incompatibility and occasionally of A B O incompatibility, hæmolysis is a major factor; but infants in whom such manifestations occurred have not been included in the series of 409 cases described in this paper. Jaundice and splenomegaly are often, but not invariably, associated with a hæmolytic process; the absence of correlation between these factors and mother-child blood group incompatibility in our series gives us, therefore, no indication of the type of anæmia. On the other hand, the rare association of a positive reaction to the Coombs test in cases of A B O incompatibility described elsewhere (Jakobowicz, Krieger and Simmons, 1948) suggests that

TABLE IIIB.
Red Cell Counts (Millions per Cubic Millimetre).

Range of Red Cell Counts. (Millions per Cubic Millimetre.)	Number of Cases.					Group Range. (Millions per Cubic Millimetre.)
	Group I.	Group II.	Group III.	Group IV.	Total.	
First day:						
>7·95	0	2	2	0	4	
7·46 to 7·95 ..	3	0	1	1	5	
6·96 to 7·45 ..	1	3	0	0	4	
6·46 to 6·95 ..	4	11	4	0	19	Group I: 7·61 to 4·76.
5·96 to 6·45 ..	39	35	17	11	102	Group II: 8·34 to 3·45.
5·46 to 5·95 ..	73	76	26	8	183	Group III: 8·85 to 4·18.
4·96 to 5·45 ..	35	18	6	9	68	Group IV: 7·79 to 4·23.
4·46 to 4·95 ..	1	5	4	1	11	
3·96 to 4·45 ..	0	1	3	1	5	
3·46 to 3·95 ..	0	1	0	0	1	
2·96 to 3·45 ..	0	1	0	0	1	
Total	156	153	63	31	403	
Third day:						
7·46 to 7·95 ..	1	0	0	0	1	
6·96 to 7·45 ..	1	1	2	0	4	
6·46 to 6·95 ..	1	10	7	3	21	Group I: 7·56 to 4·10.
5·96 to 6·45 ..	28	44	17	3	92	Group II: 7·10 to 3·97.
5·46 to 5·95 ..	63	64	24	10	161	Group III: 7·08 to 4·28.
4·96 to 5·45 ..	51	31	9	14	105	Group IV: 6·91 to 4·92.
4·46 to 4·95 ..	1	4	4	1	10	
3·96 to 4·45 ..	1	0	3	0	4	
3·46 to 3·95 ..	0	2	0	0	2	
Total	147	156	66	31	400	
Sixth week:						
5·96 to 6·45 ..	0	1	0	0	1	
5·46 to 5·95 ..	0	3	2	1	6	
4·96 to 5·45 ..	9	7	4	1	21	Group I: 4·97 to 2·95.
4·46 to 4·95 ..	3	19	7	5	34	Group II: 6·07 to 2·40.
3·96 to 4·45 ..	15	44	20	8	87	Group III: 5·64 to 2·50.
3·46 to 3·95 ..	19	54	14	8	95	Group IV: 5·53 to 2·92.
2·96 to 3·45 ..	8	25	6	4	43	
2·46 to 2·95 ..	1	2	3	1	7	
<2·46	0	1	0	0	1	
Total	55	156	56	28	295	

TABLE IV.
Incidence of Jaundice and Splenomegaly during First Week of Neo-natal Life in 336 Babies.

Relationship of Maternal and Infantile Blood Factors.	Jaundice.	Splenomegaly.	Jaundice and Splenomegaly.	No Jaundice and No Splenomegaly.	Total.
A B O compatible, Rh compatible	57 (24·8%)	22 (9·6%)	15 (6·52%)	136 (59·1%)	230
A B O incompatible, Rh compatible	17 (24·6%)	4 (5·8%)	5 (7·2%)	43 (62·3%)	69
A B O compatible, Rh incompatible	5 (16·6%)	3 (10·0%)	8 (26·7%)	14 (46·7%)	30
A B O incompatible, Rh incompatible	1	1	—	5	7

the anæmia present in such cases is not the result of fixation of antibodies, hæmolytic or otherwise, to the red cells.

It may be postulated, therefore, that there are two processes brought about by blood factor incompatibility which may act with detriment to the infant. The first of these is a process which inhibits normal utilization of iron (and possibly other hæmatopoietic substances). This process appears to predominate or to be the only one operative in cases of A B O incompatibility and in those cases of Rh incompatibility in which there are no demonstrable Rh antibodies in the maternal serum. Its effects may be so small that they are not always detectable in individual cases, but need statistical analysis of an adequate series to reveal them.

The second process is the hæmolytic one, which, when present, may give rise to such severe manifestations that the coexistent presence of the dyspoietic element is masked. If the infant survives the early fulminant effect of the hæmolytic process, the depression of hæmatopoiesis brought about by the first element may be detectable and may be, in part at least, responsible for the late anæmia.

The article by Potter and Bernstein (1948) referred to above appeared while this paper was in preparation, and it is to be noted that these authors did not demonstrate differences such as those we found between the hæmoglobin levels of babies with blood factors compatible and incompatible with the maternal factors.

In a recent paper, Mollison and Cutbush (1948) draw attention to the difference between the hæmoglobin levels of cord blood and that taken during the first few days of life, attributable to "transfusion" of placental blood. Such a difference might influence actual values obtained; but in our series standard practice was to tie the cord when pulsation had ceased; the series is therefore uniform in respect to this factor, so that comparison between our four groups is not affected by it.

It is obvious that Rh incompatibility which has not resulted in demonstrable sensitization of the mother and the large majority of cases of A B O incompatibility between mother and child give rise to clinical effects which are very mild. For this reason it seems unlikely that such incompatibilities would effect survival to a degree sufficient to suggest modification of the accepted conception of the non-adaptiveness of the blood groups—that is, their value as non-selective factors in evolution.

Summary and Conclusions.

1. A series of 409 babies has been classified into four groups (I, II, III and IV) according to the blood factor relationships between mother and child. These babies were examined on the first and third days and during the sixth week after birth.

2. In Group I (A B O and Rh factors of mother and child compatible) the mean hæmoglobin level on the first day was 151 ± 1%, this figure being equivalent to 21·14 ± 0·14 grammes per 100 millilitres of blood; on the third day 149 ± 1%, equivalent to 20·86 ± 0·14 grammes per 100 millilitres of blood; and during the sixth week 95 ± 1%, equivalent to 13·3 ± 0·14 grammes per 100 millilitres of blood.

3. In babies otherwise healthy, but with A B O (Group II) or Rh (Group III) or both these factors (Group IV) incompatible with the corresponding maternal ones, the mean hæmoglobin levels were significantly lower, the average fall being 8·5% at any of the three time intervals after birth at which they were examined.

4. There was no significant difference between the mean red cell count (5,780,000 per cubic millimetre) of babies in Group I and those of Groups II, III and IV, the mean counts in the latter groups being 5,820,000, 5,830,000 and 5,720,000 per cubic millimetre respectively.

5. As the relationship between the hæmoglobin levels and red cell counts indicates, the colour indices in all groups on the first day were above unity and were highest (1·31 ± 0·1) in Group I, the figures for Groups II, III and IV being 1·24 ± 0·01, 1·21 ± 0·02 and 1·12 ± 0·02 respectively.

6. On the third day after birth the hæmoglobin levels and red cell counts were slightly lower, but showed similar relationship.

7. Reexamination during the sixth week showed a mean hæmoglobin level of 95 ± 1% (13·3 ± 0·14 grammes per 100 millilitres) in Group I, and 85 ± 1% (11·9 ± 0·14 grammes per 100 millilitres), 88 ± 2% (12·3 ± 0·28 grammes per 100 millilitres) and 84 ± 3% (11·8 ± 0·42 grammes per 100 millilitres) in Groups II, III and IV respectively, with red cell counts in each group of approximately 4,000,000 per cubic millimetre, the colour indices being in the region of unity in each group.

8. Possible interpretations of our findings are discussed.

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CHEST DISEASES AS SEEN IN GENERAL PRACTICE.[1]

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Melbourne.

IT is to the general practitioner that most patients with acute or chronic pulmonary disease first report their symptoms, and amidst the large numbers of patients who present the most common symptoms of pulmonary disease, namely cough and sputum, it devolves on the general practitioner to effect the primary sorting into those suffering from conditions whose prognosis is serious and who require intensive investigation and treatment, and those who manifest what appear to be the normal concomitants of our modern life and the result of our minor pernicious habits and vices, for example, smoking. In the presentation of a comprehensive survey of those conditions which are most commonly seen by the practitioner, the restrictions imposed by time require the survey to be brief and succinct, yet one which may be of assistance in preliminary sorting, and may systematize the varying conditions and their symptoms. It would appear that this may best be accomplished by considering first, and very briefly, the age periods at which certain pulmonary conditions are most likely to be found, and then by considering those symptom complexes which demand serious attention by the practitioner.

The seven ages of man as painted by Shakespeare in "As You Like It" apply to thoracic diseases as closely as they do to most other ills to which the flesh is heir, and in each "age", certain pulmonary conditions are most frequently discovered.

> "At first the infant,
> Mewling and puking in the nurse's arms."

Some developmental defects in the lungs are not incompatible with life, but certain pulmonary conditions in early infancy may lead to confusion with other intrathoracic abnormalities, and of these, giant lung cysts and mediastinal emphysema may cause the infant to "mewl", but with the characteristic features of pulmonary distress, namely, cyanosis and shortness of breath.

Mediastinal emphysema may easily be overlooked, causing as it does cardiac and respiratory embarrassment, unless it is remembered that, in the child particularly, a distressing cough which may be associated with acute coryza, whooping cough, or pneumonia, may lead to intrapulmonary extravasation of air which passes along the tissue planes to the lung hilum and into the mediastinum. Air or subcutaneous emphysema above the sternum is not always present, despite an accumulation in the mediastinum of a sufficient volume of air to cause cardiac and respiratory embarrassment. Postero-anterior X-ray pictures of the chest do not always disclose its presence, and in a child with cardio-respiratory embarrassment associated with an irritable cough, lateral chest films are vitally important in diagnosis. Awareness of this condition and its prompt relief by aspiration of the air may well determine the outcome of acute respiratory infections in infants.

Lung cysts in infants are not uncommon, and when discovered, usually cause consternation to the attendant medical practitioners. The condition causes progressive dyspnœa in some infants, although at times the onset of dyspnœa may be sudden and dramatic. The cause of the dyspnœa may be difficult to detect clinically, unless there has been marked cardiac displacement when the presence of hyperresonance on the contralateral side leads to a suspicion of the presence of a complete unilateral pneumothorax, a suspicion which is strengthened by the X-ray appearances. Complete spontaneous unilateral pneumothorax is uncommon in infants, and these appearances are more commonly due to a giant lung cyst which compresses the remainder of the lung than to a pneumothorax. Although these cysts may at times be safely aspirated, there is a danger in this procedure of causing a tear in the cyst wall with the subsequent appearance of a tension pneumothorax, and the only safe procedure is removal of the cyst by a competent thoracic surgeon at the time of a thoracotomy.

Few practitioners are unaware of the acute respiratory distress caused in infants by the aspiration of large foreign bodies into the trachea. However, smaller foreign bodies may be aspirated into the bronchi with very little evidence, at the time, of any respiratory discomfort. In these cases, an infant may be brought to the consulting room with fever and general malaise, and but little cough, and that dry and irritable. An X-ray film of the chest may show only a small area of pulmonary collapse, or the appearances typical of lung abscess with a fluid level. Failure to respond to antibiotics after a very short period of time must lead to suspicion of a foreign body, or lung abscess requiring specialist medical or surgical attention.

Although other pulmonary conditions of a chronic character arise in infants, it is not until the infant reaches the stage of

> ". . . the whining school-boy, with his satchel
> And shining morning face, creeping like snail
> Unwillingly to school",

that some of the more chronic pulmonary infections of childhood become apparent, and cause symptoms.

It is during this age that two relatively common chronic pulmonary disabilities become apparent. Of these the first is asthma, often seen particularly in the child who "creeps like snail, unwillingly to school". The picture of childhood asthma is well known to you all, particularly in its relationship to recurrent upper respiratory infections, so that acute attacks often appear to be precipitated by a "cold" and the appearance of bronchitis. Prophylaxis against infections and the augmentation of natural resistance to infections by the regular administration of large doses of ascorbic acid are often of great value in control of these attacks. It must also be appreciated that foreign bodies in the lung may cause attacks of spasmodic breathing often called "asthma".

Bronchiectasis is perhaps the most common chronic respiratory disease discovered during this period. The child with a chronic "loose" cough, following an acute infection such as measles or pertussis, must be strongly suspect. Foul breath or sputum is only late in its appearance when bronchiectasis is present, and a persistent, rattling cough in a child or in a young adult must give rise to acute suspicion of the presence of this condition. Added support is given to this suspicion if, after the child is postured and has been made to expectorate into a basin containing a little water, the sputum is seen to be purulent and fairly copious in amount. Confirmation of the diagnosis is obtained by bronchography, and it is important to be aware that bronchiectasis cannot be excluded by plain postero-anterior X-ray films of the chest. Furthermore, incomplete bronchograms are of very little value, for it is of vital importance to the physician or surgeon in charge of the patient to know the exact distribution of bronchiectatic pulmonary segments, and the extent of pulmonary involvement. In regard to treatment of this condition, it must be categorically stated that the only satisfactory method of treatment is the surgical removal of involved areas. The development of the technique of "segmental resection" has allowed many children and young adults to be given the advantage of surgery in this condition, as we are aware that the lung tissue continues to develop until early adult life, and therefore more pathological lung tissue can be removed in childhood than in adult life. Early diagnosis of bronchiectasis in childhood is, therefore, of vital importance, and it is to the general practitioner that we look for detection of this condition.

During this phase, the spectre of pulmonary tuberculosis also rears its ugly head. In childhood tuberculous infections of the lungs the classical signs and symptoms of adult tuberculosis are not seen. However, there is invariably some suggestion of direct contact with a patient suffering from open tuberculosis, and the diagnosis is confirmed by X-ray evidence of pulmonary disease, an associated positive Mantoux reaction, and the presence of tubercle bacilli in the fasting gastric content. The most common manifestation of pulmonary tuberculosis in childhood is the so-called "primary complex", comprising a shadow in the peripheral lung field with associated hilar adenitis. The vast majority of these lesions will resolve with a simple régime of rest, fresh air, and an adequate diet, and the use of streptomycin in these cases is not favoured.

Although other chronic pulmonary diseases are not unknown in childhood, for example the pulmonary condition associated with congenital cystic disease of the pancreas, they are uncommon, and their bizarre character and the peculiar nature of the X-ray film of the chest almost invariably lead to meticulous study in an attempt to elucidate the problem. It does not, therefore, appear to warrant description in this lecture of such conditions.

> ". . . the lover,
> Sighing like furnace, with a woeful ballad
> Made to his mistress' eyebrow",

[1] Read at the Melbourne Post-Graduate Committee's Refresher Course, Ballarat, on February 19, 1949.

may well represent the adult asthmatic, excitable, "sighing like furnace", and often of rather unstable temperament. However, asthma appearing first in adult life may be primarily caused by specific pulmonary infections. Bronchial tuberculosis, bronchiectasis of restricted distribution, or a small bronchial tumour of the "adenoma" type may be the primary cause of these "asthmatic" attacks. Even in the absence of any significantly abnormal X-ray appearances in the lungs, the presence of an asthmatic wheeze demands an examination of the sputum, both for tubercle bacilli and for other pathogenic organisms.

Adenoma of the bronchus is a tumour arising in the bronchial wall, and commonly seen in this age group. Its characteristic features are its localized nature, and the fact that it is most commonly found in the main stem bronchi. Because of its character and nature of growth, the symptoms it produces are either those of hæmorrhage from its surface or of obstruction to the bronchus. The hæmorrhage may be slight and consist only of streaking of the sputum, and there may be only one incident of this nature in a period of many years. Bronchial obstruction leads to the characteristic diagnosis of recurrent pneumonia. Any patient who has had signs and symptoms of "recurrent lobar" pneumonia may, in fact, have a bronchial tumour. Because of its serious nature, and despite the fact that the "bronchial adenoma" is regarded by many as being benign in character, there is considerable evidence that this tumour is, in fact, a slowly growing malignant tumour, and, therefore, early diagnosis and satisfactory treatment by resection are of vital importance.

> "Then a soldier,
> Full of strange oaths, and bearded like the pard,
> Jealous in honour, sudden and quick in quarrel,
> Seeking the bubble reputation
> Even in the cannon's mouth",

may well be stimulated by the toxæmia of pulmonary tuberculosis. The old descriptions of lassitude, loss of energy and loss of weight, with cough and sputum, whilst being those symptoms most commonly seen in the adult, may be masked in some by a hyperaggressiveness, and often, by heightened or by decreased sexual activity. Awareness of pulmonary tuberculosis as the commonest chronic pulmonary infection in the adult has been impressed on all practitioners, and any individual with or without symptoms should not be regarded in our modern day and times as having had a complete medical examination if an X-ray examination of his chest has not been made. The discovery of abnormal pulmonary shadows in an X-ray film of the chest must indicate that tuberculous infection should be excluded. Pulmonary tuberculosis does masquerade in many guises—as pneumonia, lung abscess, chronic bronchitis, tumours *et cetera*, and when an abnormal X-ray shadow is discovered, a study of the sputum for tubercle bacilli is mandatory.

It is also at this period of a man's life that the effects of certain occupational hazards may be seen. Although silicosis and silico-tuberculosis of mining are the commonest of these, and lead to the symptoms of cough and dyspnœa often regarded as "bronchitis", many other industries may lead to pulmonary disability due to the inhalation of dusts. Of these I would mention only boiler-scaler's disease due to iron salts, asbestosis, anthracosis and the peculiar and often progressive pulmonary disease associated with the inhalation of beryllium dust. These conditions often resemble in their clinical and radiological characteristics the peculiar condition of sarcoidosis and also the commoner condition of pulmonary tuberculosis.

Non-parasitic lung cysts of varying nature and ætiology may be discovered during this period of life either as the result of mass radiography or because of symptoms arising from infection of the cyst. When the diagnosis is suggested by this infection, it is not uncommon for a diagnosis of "lung abscess" or "encysted empyema" to be made, and an error of this nature may lead to chronic invalidity of a patient whose condition can be cured only by resection of pulmonary tissue. Drainage of infected lung cysts is never satisfactory, almost invariably leading to chronic draining sinuses on the chest wall, and mutilating and ineffective secondary operations.

Over the

> ". . . justice,
> In fair round belly with good capon lin'd
> With eyes severe and beard of formal cut"

the solemn spectre of a pulmonary carcinoma casts its shadow. It is in the age-group of forty to sixty years that this increasingly common condition is most frequently discovered, and if recognized sufficiently early in its course, it is the form of malignant disease most amenable to surgery. Awareness of the increasing incidence of this condition and of its apparently symptomless onset is the greatest single aid to its early diagnosis, and a routine X-ray examination of the chest in this age-group and the careful investigation of any abnormal pulmonary shadow will be the greatest forward step in obtaining the earlier treatment of carcinoma of the lung.

The appearance of a "lung abscess" or of "unresolved pneumonia" in a patient of this age, must raise an immediate suspicion of carcinoma. A single localized shadow in the lung periphery or "enlargement of one hilum" in the X-ray film of the chest, even in the absence of specific symptoms referable to the lungs, may be the first indication of the presence of a carcinoma of the bronchus.

I would here suggest that exploratory thoracotomy on the suspicion of a carcinoma of the lung is as completely justifiable as an exploratory laparotomy for an undiagnosed abdominal condition, and that this procedure, in the hands of a competent thoracic surgeon, is just as safe as is an exploratory laparotomy by a general surgeon.

> "The sixth age shifts
> Into the lean and slipper'd pantaloon,
> With spectacles on nose and pouch on side;
> His youthful hose well sav'd, a world too wide
> For his shrunk shank; and his big manly voice
> Turning again toward childish treble, . . ."

In this period of sere and yellowing age, perhaps the commonest chronic pulmonary condition is pulmonary tuberculosis. In this age group, the infection appears relatively benign and may produce very few symptoms other than chronic cough, but by virtue of the infective nature of the sputum, may constitute a grave menace to associates and relatives, and particularly to infant contacts. It is, therefore, never safe to regard "grandfather's bronchitis" as a benign symptom of increasing years and care must be taken to exclude the dangerously infective "elderly tuberculosis".

In the

> "Last scene of all,
> That ends this strange eventful history",

we see the appearance of cardiac asthma, or orthostatic pulmonary congestion, subsequent to the aging of tissues, pulmonary as well as cardiac, and their slowly progressive cessation of function.

In this brief and cursory review of some of the varying conditions which affect the human pulmonary tissues through the course of years, no mention has been made of a condition, most commonly diagnosed clinically, but rarely recognizable as a pathological entity. I refer to "bronchitis", which is diagnosed at all ages of life. Whilst believing that such a condition does exist, I would also consider that this diagnosis should be made only when other recognizable pathological conditions have been excluded, and full recognition has been given to the possible effect of chronic antral sinusitis in its symptomatology. Many or all of the conditions so far described may be, at some time, described as causing "bronchitis", which is only a symptom complex, and I would, therefore, emphasize the importance of exclusion of these conditions before one accepts a final diagnosis of "chronic bronchitis" as being the origin of symptoms. Similar comments may be made regarding "unresolved pneumonia", for there must always be a cause of non-resolution, and this cause must be diligently sought.

Certain specific symptoms throughout all age groups demand close investigation. Of these, the commonest is cough and sputum, and any practitioner may in these days be regarded as culpable unless these two symptoms are investigated by means of an X-ray examination of the chest and a sputum examination for the prevailing organisms.

Hæmoptysis, or blood-streaked sputum, must always be regarded as of serious import, and a simple explanation of this symptom as due to "retropharyngeal varices" must be accepted only after all other causes have been excluded. A survey of the various conditions which may produce hæmoptysis covers bronchiectasis, lung abscess, bronchial adenoma, carcinoma of the lung, and pulmonary tuberculosis, among many others. Exclusion of these conditions by X-ray examination, sputum examinations and bronchoscopy is almost invariably required, and I would here plead for the wider use of bronchoscopy as a

diagnostic procedure, and would suggest that this procedure is one which should be carried out by physicians.

In conclusion, I should like to reiterate that the greatest responsibility in the preliminary sorting of all these cases does devolve on the general practitioner. His responsibilities are great, because he sees all these patients first, and must often rely on the reports of specialists concerned with inanimate findings such as those of X-ray examinations. In the sphere of chest diseases, the personal liaison between the practitioner, radiologist and clinical pathologist must be intimate, and both specialists must be closely in touch with the nature of each individual problem presented to the practitioner by each patient referred to them for study. If this liaison becomes sufficiently close, I feel certain that the problems in chest diseases which we face at present, and which are largely due to delays in diagnosis rather than to the low efficacy of our forms of therapy, will be largely overcome. Awareness of the various conditions and a constant search when they are suspect are, I feel, the crucial responsibility of the general practitioner.

TRAGEDY AT GREEN PONDS, VAN DIEMEN'S LAND, 1842.[1]

By W. E. L. H. CROWTHER,
Hobart.

VERY soon after the first World War I had to attend an inquest at Brighton, a little township at the crossing of the River Jordan, some eighteen miles from Hobart. It was held at the local courthouse, a one-story freestone building, which dated from the early years of the settlement of the colony. In the office, on table and desk, were the usual charge books and correspondence, while shelves around the walls and tops of cupboards were piled high with small bundles of papers, tied with pink tape and faded with the sun—records of vital interest in their time and long since forgotten.

Whilst waiting to be called, I took some down and commenced to look through them. Remembering that Jorgen Jorgensen, self-styled King of Iceland, ex-convict and author, had been a member of the field police of the adjoining district, I hoped to see his even, well-formed writing. Not finding it, I refolded the stained and dusty pages and returned them to their places. Within a few years the old courthouse and walled gaol, with the inn at the bridge, were demolished and the unspeakable new police buildings built. The records were burned except for a charge book or so and a number of documents, taken by those who were present at the time. More years were to pass before, one day in my surgery, some of these papers were offered to me. After looking them over I bought a file of unusual interest. It was the original transcript of evidence as to the cause of death of Mrs. Jane Clements, of Green Ponds, written with a broad quill pen on the hand-made, watermarked paper of the period. The inquest had been held there by the coroner, Mr. G. B. Forster, two days after her death, on March 14, 1842. She had died after a difficult and protracted labour, with, it was alleged, neglect by her medical attendant.

The little settlement of Green Ponds (formerly Green Water Ponds) and today Kempton, then, as now, extended for a mile or more on either side of the main road, about midway between the top of Constitution Hill and the Cross Marsh. It included two inns, the Royal Oak and the Good Woman, and Mr. Ellis's store, with a blacksmith's forge and cottages for those who served the coaches and teams which passed each day to and from Hobart Town and Launceston. Otherwise the community depended on such large estates as Mount Vernon, Hunting Grounds and The Wilderness with the small farms of its fertile valley. At this time there was the Picton road party in the vicinity and some kind of a probation station. Two of our profession resided in the township: Dr. D. Stodart,[1] who had been posted as district assistant surgeon in 1841, and Dr. T.G.,[2] who had lived there for nearly twenty years following an appointment in 1823 as surgeon to the road gangs. As many of his descendants still live in Tasmania and may not wish the part he played to be recalled, initials have been used throughout this narrative rather than his proper name. The old doctor must have been a considerable figure in his community and apparently helped out his meagre salary by acting as the local brewer. "Woodville", his home, has long been demolished; but members of his family lived at "Fernleigh", just across the main road, until about five years ago.

The verdict of the coroner is not known. The file appears to be complete, but the result of the inquest is not recorded on it. A long and unproductive search for additional information was made in the contemporary newspapers — *Hobart Town Advertiser*, *Hobart Town Courier*, *Colonial Times*, *Tasmanian and Australasiatic Review* as well as in the Launceston Press, but apparently the fatality did not come to the notice of the reporters concerned.

Here is the evidence as transcribed. Those who have had to conduct such labours may decide whether or not any charge of neglect should be sustained.

The Evidence.

George Clements being duly sworn, states as follows: The deceased Jane Clements aged 34 years was my wife. She was taken ill on Saturday the 5th inst. at about 7 o'clock in the evening—she was in labour—her pains came on very sharp and quick at about 9 o'clock the same evening. By my wife's desire I went for Mr. G., Surgeon, the regular medical attendant of my family, who arrived in three-quarters of an hour. He saw the patient (now deceased) and made her change her position to that of lying on her left side, her knees bent up—and directed her to place her feet against the bed post—her head was about the centre of the bed.

Mr. G. stated to me that as sickness had taken place (meaning vomiting) that generally under such circumstances it was a symptom of lingering labour—after this—when he had been in the house about half an hour he went to sleep, having previously drank a glass of Brandy and water, this was a little before 10 o'clock in the evening. During the time Mr. G. slept, the deceased's pains increased for full an hour and a half—when she said to me in one of her pains "see that Old Beast sitting asleep when he might be helping me in my pains. I am sure my pains are strong enough and my child ought to be born". There were present at this time Ann McDonald, assigned servant to Mr. Smailes and Mrs. Emma McVilly, wife to Mr. Richd McVilly.

I asked one of the before mentioned women to awake Mr. G., which she did. He came to the deceased, and looked into her face. He said in my hearing, during one of her pains: You must have patience my good Woman; patience is all you need. There is no danger; time is all you require.

Mr. G. then went to sleep in the chair from which he had got up. He went to sleep without having touched my wife (the deceased), but only having looked into her face. The floor of the room in which the deceased lay was sunk below the surface—two steps—and not floored or paved. My feet were very cold standing on the earth. The deceased's pains continued with very little intermission until an hour before daylight, when I awoke Mr. G. with my own hand, and I swear that he was fast asleep. I asked him whether it was not necessary to use warm fomentations, and without getting off his seat he said: No; you can do her no good to force her labour, but you may force a Cold. The deceased took nothing from the time that Mr. G. arrived until this time. I then asked whether I might not administer a little some-

[1] Read at a meeting of Tasmanian Fellows and Members of The Royal Australasian College of Physicians, Launceston, September 25, 1948.

[1] Dr. D. E. Stodart arrived at Van Diemen's Land by the *Minerva* in 1822. He went back to England about 1834 and graduated at Edinburgh. On his return to the colony the following notice appeared in the *Hobart Town Advertiser* of November 17, 1840: "Card. D. E. Stodart, M.D., begs to inform his friends and the public that his present residence is No. 62 Macquarie Street, where he will be happy to attend all professional calling. N.B.: Dr. Stodart intends practising as an *Accoucheur*, having particularly attended and obtained a Diploma in that branch of his profession."

[2] Dr. Thomas G. arrived with his wife and two children at Hobart Town by the *Surrey* on August 26, 1820. He was appointed attendant, road gangs, Green Ponds, on November 15, 1823. A licence to brew at his premises was granted on March 29, 1824. His brother Francis was catechist in the same district. He published a medical pamphlet (*Guardian*, June 1, 1850) and died at Green Ponds on March 29, 1858.

thing to the deceased, and he said it was not necessary, but that I might give her a little Brandy if I pleased, which I did immediately.

Mr. G. prescribed the quantity—half a wineglass—I administered it with warm water and sugar which the deceased drank. Mr. G. continued on his seat and apparently went to sleep without going to the deceased. The pains continued as they had before been—from every three to five minutes without intermission—until an hour or an hour and a half after day light, when having been awoken for the third time by—(Ann McDonald)—he went to the deceased. Mrs. Emma McVilly and Caroline Beaton were present, the latter of whom had arrived about an hour previously. The pains of the deceased continued until about 11 o'clock A.M. of Sunday the 6th of this present month, and tho' I was not present the whole of the time, I was not absent more than a few minutes together.

At this time I observed that the Brandy bottle which I had left on the side table in the front room was empty, which had contained our pint on Saturday evening. I then asked Mr. G. whether I might give the deceased a little wine, there being no more Brandy. He said yes, which I administered as warm Negus. I then went to Mrs. Bessier's (Good Woman Inn) and got another pint of Brandy, which I placed on the same side table as before. Between this and 1 o'clock Mr. G. frequently went into the bedroom where the deceased was, her pains had become more lingering—and less active—and at this time he went away to his dinner and returned about 2 o'clock. He then went into the bedroom and looked at the deceased; he afterwards sat down by the fire. About 20 minutes afterwards he laid down on the sofa and remarked "that it was no use fatiguing himself then—for that he might be exhausted at the time when he would be most wanted".

He then apparently went to sleep. At nearly 5 o'clock he rose and came to the deceased who still continued in the same state of lingering labour and very much exhausted. He put his hand under the bedclothes and feeling her person he directed her to change her position and lie on her back. The deceased tried to do so, which after some difficulty she accomplished. She remained in this position from about 5 o'clock to 7 p.m. Her pains increased in violence, but at the same intervals. During these two hours Mr. G. never came to her. At 7 o'clock she changed her position to her left side with my assistance. Mr. G. went to her. He came out and said that the pains had increased; the child was alive and fair (?) for the world. He went in and out of the bedroom where the deceased was until about half past eight o'clock when he again seated himself and apparently went to sleep. Previous to this I had observed that the brandy bottle was empty. At about 10 o'clock P.M. Mr. G. again visited the patient, and he returned informing me that it would be a lingering case, but without danger. He then returned to his chair as before, and I laid myself on the table and went to sleep, leaving my wife in charge of Caroline Beaton. I was awake [*sic*] (at what hour I do not know) by the screams of my wife (the deceased). I found Caroline Beaton and Mr. G. both asleep, the deceased said she had had two pains since I went to sleep. I called out Dr. G.! And he came. He was in and out of the bedroom from that time until 7 o'clock on Monday morning when he said as the pains were decreased he would go home and refresh himself, instructing me to place the deceased on her back, and to call him if the pains increased. His residence is about three quarters of a mile from my house by the way he came. She was placed on her back accordingly and turned on her side again between 9 and 10 o'clock. At 11 o'clock in the forenoon of this day Monday the deceased was in great pain, and I went for Mr. G. He without delay came and passed me on the road, and went to the deceased. I found him with her when I arrived at nearly 12 o'clock noon. Mr. John Franks came to ask Mr. G. to see a case of accident which had happened to one of his men if there was no danger in leaving the deceased, as he could not find any other medical man. Mr. G. expressed it as his opinion that the deceased was not in any danger and that he would go and see the other man in question and return to his own house (which was in the way), instructing me to come to his house if necessity required it. At about 2 o'clock p.m. the pains of the deceased became more active and authorised my going immediately for Mr. G. He came immediately and brought his surgical instruments. He went and looked at my wife (the deceased) and reported favourably and returned again to his dinner at his own house. At half past three o'clock Mrs. Wellington, a midwife, came in. She saw and examined and felt the deceased. She disapproved of her lying on her back and made her lay on her left side. She administered about half a wineglass of Gin or Hollands and warm water and applied a warm flannel; and by Mrs. Wellington's further directions the deceased was taken out of her bed and walked about the room for about 10 minutes. Her pains increasing, this was repeated two or three times until Mr. G. came (uncalled for) between 4 and 5 p.m. Mr. G. approved of what had been done by Mrs. Wellington. Mr. G. left my house between 5 and 6 o'clock p.m., telling me that he intended to sleep at Mr. Flexmore's, very near my house, where he could be found if wanted. The deceased's pains becoming much more active two hours before daylight, I called Mr. G., who came and sat at the foot of the bed very attentively timing the labours of the deceased. He continued this watchfulness for about an hour and then fell asleep by the fire. In another hour I went at the request of the deceased and called Mr. Stodart, who immediately came, and on his arrival awoke Mr. G., who was asleep. He saw the deceased. He said he considered her in great danger and that an operation was necessary. Under the circumstances I considered it necessary to call in a third medical man (Mr. Price, dispenser of the Picton Road Party) before the operation was commenced, but finding that delay was unfavourable, I sanctioned Mr. G. and Mr. Stodart doing whatever in their judgement was necessary. After this Dr. Stodart urged me to go or send for Mr. Price and tell him to bring all his instruments, for that life depended on his speed. This was about half past six o'clock a.m. of Tuesday the 8th. I returned with Mr. Price about 7 o'clock.

Questioned by the Jury: I did not see any person drink the Brandy which I missed out of the Brandy Bottle, or did I partake of any of it myself. It was accessible to everybody who came into the house. Mrs. Wellington brought the Gin and Hollands with her. I have known Mr. G. for twelve months. It did not appear to me that Mr. G. was at any time tipsy or affected with liquor. He could not get at the Brandy bottle without getting off his seat near the fire and proceeding two steps. My ground of suspicion that the deceased lost her life by neglect of Mr. G. proceeds from the circumstance of Mr. Stodart having stated to me that the deceased on his arrival was in great danger, and Mr. G. at the time being asleep, I considered that the patient was neglected.

Given before me on this 14 day of March, 1842, at Green Ponds. GEORGE CLEMENTS.

G. B. FORSTER, Coroner.

The inquest was now adjourned, and on its resumption the first witness called was Mr. David Edmund Stodart, who, being duly sworn, made the following statement:

I was called upon about 5 o'clock on the morning of the 8th inst. Tuesday by Mr. Clements to attend his wife, the deceased. I immediately accompanied him to his house. On my arrival I found Mr. G. there. He was sitting by the corner of the fireplace, and he arose as I entered the room. I did not awake him, or was I aware that he had been asleep. I conversed with Mr. G. for a few minutes and then we both went into the bedroom of the late Mrs. Clements, the deceased. I found her, considering the lingering labour she had endured, better and stronger than might have been expected. I told Mr. Clements that she was in danger and that it might be necessary to have recourse to an operation, but I did not observe nor had I any reason to suppose neglect or professional insufficiency in the medical person who was in attendance before me.

The cause of the difficulty was most evident. The child's head was too large to pass through the pelvis of the mother. Mr. Price was sent for, who was very favourable in his opinion, but the labour being still lingering and nature having performed the whole of its part, we were obliged to have recourse to operative means, which was done, and the child extracted. We were of the opinion that the deceased was in great danger. Inflammation ensued, and in spite of every effort either of skill or attention on the part of the medical attendance the deceased died on Saturday morning at between 8 and 9 o'clock a.m., the 12 of the present month. Mr. G. was not in the least in liquor at the time, and though I saw some Brandy produced in a bottle, and I saw Mr. G. drink about half a wine glass, and two thirds of a wine glass was administered to the deceased. A short time afterwards when I should have been glad of some myself I found that it was nearly all gone and certainly not by Mr. G.'s drinking; it appeared to me that it was open to every person who came in. Mr. G. appeared very much fatigued, and I offered to take charge of the deceased during the night of Tuesday.

Given before me this 14 day of March at Green Ponds. D. ED. STODART, M.D.

G. B. FORSTER. Coroner.

Thomas Law Price being sworn states: I am the Dispenser of the Picton Road Party. Last Tuesday morning the 8th

of the present month I was called upon by Mr. Clements at between 8 and 10 o'clock a.m. to attend his wife (the deceased). I went with him to his house. He asked me to bring my instruments, but as I ascertained that there were instruments already there I did not take mine. When I arrived Mr. G. and Mr. Stodart were in attendance upon the deceased. I at first consulted with these gentleman, and then I examined the deceased. I found that the labour was protracted. I again spoke to Mr. Stodart and Mr. G. and medical means was administered with partial success. After some little time it became quite requisite to use the instruments to abstract the child. Mr. Stodart and myself operated and without success, which after the lapse of some little time the child was brought into the world by us. It was dead, and the deceased ultimately died of inflammation. I depose that at the time I first saw the deceased on this occasion the state I found her in might have occurred from natural causes, in spite of the aid of all the medical men in the Colony.

Sworn before me at
Green Ponds this 14
day of March, 1842. THOS. L. PRICE.
G. B. FORSTER,
Coroner.

Comment.

These are the facts as presented to the coroner, and it is clear that none support the charges of neglect. Indeed, Mr. Forster might well have commended the humanity and patience shown by Dr. G. during his long vigil at the bedside. Few if any of our generation and training would have remained with their patient from Saturday evening to the forenoon of Tuesday, with only an hour or two on Sunday and Monday and part of that night away from her home. The strain of the constant attendance in the sick-room and contact with anxious relatives must have been almost unendurable.

If there was no neglect on the part of Dr. G., was any grave error in his judgement responsible for the loss of mother and child? Strangely little is revealed of the patient herself. There is so much we cannot know a century after the event. Was this her first labour? Had the membranes ruptured before its onset? If not, when did they do so? Again, was there disparity between the fœtal head and the mother's pelvis, as stated by Dr. Stodart, or, as the attitude of Dr. G. suggests, primary uterine inertia or an abnormal presentation? Had Dr. G. elected to give evidence much would have been made clear to us. It is obvious that the labour was abnormal from its start and he was faced with a patient who had taken to her bed with the onset of pains and remained there. In retrospect, Mrs. Clements should have been kept about her simpler household work for part at least of the first stage, to aid uterine action.

What other action could Dr. G. have taken to safeguard his patient and her child? General anæsthesia was still to be discovered, and to have used even a sedative at this period would have been to run counter to Biblical tradition. That there were sedatives capable of lessening pain and resting a patient may be learnt from Phillips's translation of the "*Pharmacopœia Londinensis*" of 1836. Morphine (discovered in 1803) was then official in the forms of the acetate and hydrochlorate, and opium was available as confection, extract, tincture and enema. If a physician had to select one of these, laudanum (*Tinctura Opii*) would almost certainly be his choice. The labour of the time was conducted with a minimum of exposure of the patient, and while it is stated that Dr. G. put his hand under the bedclothes and felt her person, it is not certain that he actually examined her *per vaginam*. Against this, Mrs. Wellington, the midwife, had examined and felt the patient a little over an hour previously and no doubt informed him of her findings. It is evident that there was not sufficient progress to allow the use of instruments, which he had brought to the house earlier in the afternoon, and he played the strong card of delaying an attempt at delivery. Fifteen hours later, after a consultation, Mr. Price states, it was decided to try medical means (which were employed with "partial success") before resorting to instrumental delivery. It will be remembered that this attempt was unsuccessful, but after a "little time" a dead child was "brought into the world by us". It is not stated if perforation with the use of the dread cephalotribe and cranioclast was performed; but the subsequent death of the mother from inflammation suggests that severe trauma was associated with the delivery.

After consideration of all these factors it is possible that the inactivity of Dr. G., who was an experienced practitioner, was the correct procedure. Eventually, with rupture of the membranes, moulding might have permitted some descent of the presenting part and delivery of the child, even if it was stillborn.

Looking back, we can only wonder at the patience, suffering, courage and humanity displayed by those concerned in this tragic happening of over a century ago.

SURGERY OF THE COMMON BILE DUCT.[1]

By B. T. EDYE,
Sydney.

BEFORE opening the discussion I wish to thank Dr. Hurley for his instructive paper, which is the more valuable as it traverses his personal experience in the management of cholelithiasis.

My objects are to discuss some aspects of the problem of stone in the common duct and to mention certain avoidable causes of the persistence or recurrence of symptoms following operations upon the gall-bladder.

For the purposes of comparison I have looked up the case histories of a number of patients suffering from gall-bladder disease who came under Sir Alexander MacCormick's care while I was his house surgeon at the Royal Prince Alfred Hospital in the year 1911. At that time the diagnosis depended on clinical evidence only and was often in doubt until laparotomy revealed the true pathological condition. Now with the aid of ancillary methods of investigation, it is possible to make an accurate diagnosis in all but a small percentage of cases. However, mistakes occur even at this time because sufficient use is not made of these special methods. I consider it advisable in all cases of chronic dyspepsia, at least, to order Graham's test as well as a radiological examination of the gastro-intestinal tract. An opaque meal may show deformity of the duodenal cap interpreted as being due to ulcer, while in fact the cause is distortion due to a diseased gall-bladder. On a number of occasions I have seen patients who for months or years had been treated for duodenal ulcer on the evidence of the opaque meal, and ultimately the real cause was revealed by Graham's test. A fractional test meal and a blood count may be of value in the differential diagnosis. If a stone is present in the common duct for any length of time, even in the absence of jaundice, the patient's general condition may deteriorate to such an extent as to suggest carcinoma of the pancreas, stomach or other organ. Here the difficulty is increased, as Graham's test is of no value in the presence of obstructive jaundice, and the final diagnosis may be determined only at operation. It is not generally understood that a stone may be present in the common duct for months or years without causing obvious jaundice until an attack betrays its presence. I have recently removed a stone after it had been nine years in the common duct and another after twelve years. In the year 1911 biochemical and liver function tests were not in use and the anæsthetic agents consisted of ether and sometimes chloroform, both deleterious to the liver. However, in spite of these disabilities results of surgery as performed by Sir Alexander MacCormick were extremely good. The technique he employed both for cholecystectomy and choledolithotomy differs in only a few directions from that employed by most surgeons today. Indeed modern advances in the surgery of the gall-bladder depend not so much upon improvements in surgical technique as upon the progress in diagnostic methods, the investigations of

[1] Read at a meeting of the Section of Surgery, Australasian Medical Congress (British Medical Association), Sixth Session, Perth, August, 1948.

liver function, better pre-operative preparation and post-operative care, progress in anæsthesia and chemotherapy.

The surgical methods I have practised are those of Sir Alexander MacCormick, and I have developed the technique with the object of avoiding a return of symptoms and reducing to zero the risk of injury to the common or hepatic ducts, or to any blood vessel, especially the right hepatic artery.

The incision depends upon the build of the patient, whether spare and narrow or stout and broad, and varies from a paramedian with splitting or displacement of the rectus muscle to a modified Mayo Robson or a Kocher type. If the diagnosis is in doubt a paramedian incision is used to allow access to the stomach or other adjacent organs should this be necessary. A word of warning concerning the Kocher incision—it should not be made too close to the costal margin. Some of the worst incisional herniæ I have encountered have followed this incision, and the repair has been rendered exceedingly difficult because of the proximity of the costal margin.

The gall-bladder rest or sandbag, if used, is raised to such a height as not to subject the patient's back to excessive strain. There seldom will be complaint of post-operative backache if this precaution is observed.

The gall-bladder is aspirated, if possible, and for this purpose I use an aspirator fitted with a stop-cock, such as is provided with a Potain outfit. The cystic duct is isolated and a ligature is passed around it, but not tied. In a few difficult cases, towards the end of the dissection, I have found this ligature encircling the common and not the cystic duct. The cystic artery is next displayed well on the gall-bladder to avoid the right hepatic artery and is ligated. If these structures cannot be clearly seen the attempt to expose them is abandoned. The dissection is now carried on from the fundus, careful watch being made for any abnormal arrangement, especially when the cystic duct is approached. Sometimes an accessory duct enters the gall-bladder midway between the fundus and the cystic duct. In one such instance the passage of a probe demonstrated a communication with the hepatic duct in the portal fissure. Usually, however, an accessory duct joins the cystic duct or the gall-bladder near by. Ligation of these ducts appears to do no harm, and if they are overlooked there may be an escape of bile only for a few days. The right hepatic duct may unite with the cystic duct and so be endangered. A watch also is kept for any unusually placed blood vessel, especially the right hepatic artery, which may be ligated in the belief that it is the cystic artery.

With the gall-bladder as a tractor the cystic duct is followed until its termination in the common duct is clearly defined. I have on several occasions removed a remnant of cystic duct with stones or grit which was responsible for recurrent or persistent symptoms following cholecystectomy. In one patient, one of three stones had escaped from such a remnant into the common duct and caused jaundice many years after the original operation.

At this stage it is appropriate to make some remarks in regard to the management of acute cholecystitis. It is seldom that I have found it necessary to operate as a matter of urgency. I prefer to wait until the acute symptoms have abated, the bowels have acted and the patient is able to take food. This may be a matter of days. I do not believe it is necessary to wait for months. If the symptoms are not subsiding cholecystostomy is performed, with the least disturbance of the gall-bladder for cholecystectomy may be necessary later. As a result of waiting for the symptoms to subside, cholecystectomy becomes possible and thereby the patient is saved the necessity of a second operation.

If, after consideration, it is proposed to explore the common duct, the cystic duct is ligated well away from the common duct to prevent any leakage from the gall-bladder. The cystic duct is then opened about half an inch from the common duct and a probe is passed to the duodenum and followed by sounds, according to the calibre of the duct. This may be sufficient to show that the duct is clear. If there is doubt, or if stones have been detected in the common duct, the cystic duct is slit down into it or a separate vertical opening is made at a lower level and the duct is thoroughly explored and cleared of stones. I have seldom seen the common duct so dilated that it could be explored with the finger. If only the cystic duct is opened it is ligated close to the common duct and the gall-bladder is removed. The bare areas are oversewn and the wound is closed, drainage often being omitted. If the common duct is opened it is sutured with fine catgut and a tube is placed down to the duct and brought out through a stab wound to the right, or in certain circumstances it is brought through the wound. If bile escapes the tube is kept in position until it ceases to drain. The tube is then shortened and removed on the following day. If there is no escape of bile the tube is removed after three or four days.

Where the method of closure of the common duct is used it is important to be confident that there is a free passage for the bile into the duodenum. The best method of establishing this is by the passage of sounds, such as Liston's or Hegar's dilators. A probe is passed followed by sounds in increasing sizes. The first sounds may pass easily, but soon a sense of resistance at Vater's papilla is experienced, which is overcome by gently increasing the pressure. The sounds of smaller sizes may by-pass a stone, but the larger ones will push it on and fail to engage the duct orifice; this is the signal for a further search for stones with duct forceps or scoops. If any are removed the larger sounds usually pass easily. This process is repeated until the largest sounds the duct will accommodate pass. During these manipulations no undue force is used, otherwise the duodenal wall may be torn or a false passage made.

If a stone is impacted in Vater's papilla and cannot be dislodged, its removal transduodenally may be necessary. A sound is passed down to the stone and held by an assistant so as to cause the region of Vater's papilla to project forward; the anterior wall of the duodenum is then opened transversely and an incision is made over the stone to secure its release, after which larger sounds are passed from the original opening in the common duct to the duodenum. The incision in the anterior wall of the duodenum is closed with fine chromicized gut in two layers. The transverse incision in the duodenum is used to avoid bleeding. Opening the duodenum has an additional advantage in that it has allowed the detection on more than one occasion of a carcinoma of Vater's papilla. In 1925 I detected a carcinoma in this way and was able to excise it at a later operation. In an old lady recently there was not only an impacted stone but a small carcinoma also.

Although most stones are found in the lower portion of the common duct, occasionally one or more have moved into the hepatic duct, especially where there is much dilatation. The hepatic duct should therefore be palpated and explored. Unfortunately a stone may not be detected, or it may be pushed up out of reach when search is made with the instruments; later, if small enough, it may escape through the dilated Vater's papilla or it may become arrested and cause a renewal of symptoms.

Stricture of the common duct, whatever the cause, is a distressing event, and its correction is notoriously difficult and may be unattainable. In a difficult, shrunken and adherent gall-bladder, where the anatomy is obscured and the common duct endangered, I frequently cut away the greater part of the gall-bladder, search for the cystic duct with a probe, and lay it open down to the common duct; after this the hepatic duct can be located and the separation of the remainder of the gall-bladder completed with safety. Caution is also necessary when one is dealing with the so-called "easy gall-bladder", for in my experience the story is usually that the gall-bladder was an easy one to remove and nothing untoward happened during the operation. If there is the least suspicion that a duct has been injured, often raised by an unexpected escape of bile, a careful immediate review should be instituted and wishful thinking not encouraged. If an injury is discovered, the first step is to isolate the parts by dissection and to demonstrate their relationships by the passage of sounds. If it has not already been done it is important to show that there is a free passage distally to

the duodenum. Any defect is accurately sutured, mucosa to mucosa, and to avoid tension it may be necessary to mobilize the duodenum and head of the pancreas. The suturing may be performed over a tube or catheter, the lower end of which is passed into the duodenum, or the tube may be brought externally through a separate vertical incision in the common duct below the level of suture. If the plan outlined above is followed, what a measure of anxiety and suffering for all concerned may be prevented!

If the fear of damage is not raised until later, because of the advent of jaundice or the development of a biliary fistula, the earlier plans are made to operate at an opportune time, the better. Prolonged delay in the presence of obstructive jaundice results in biliary cirrhosis with enlargement and induration of the liver. The approach during a subsequent operation is thus hampered, and there is no operation which calls for greater patience or more meticulous dissection. The recognition of the parts may be difficult and hæmorrhage may be troublesome. Before any structure resembling a duct is incised it should be explored with a hypodermic needle.

TABLE I.
Total Operations: Nine Years from January 1, 1939, to December 13, 1947.

Type of Operation.	Total of Operations.	Deaths.	Percentage.
Cholecystectomy	719	12	1·7
Cholecystectomy with exploration of common duct	458	12	2·6
Cholecystostomy	8	0	0
Total	1185	24	2·0

Many methods of repair have been used. I think my best results have followed an end-to-end anastomosis, particularly when performed early. I once used a vitallium tube, but it became blocked later with a return of jaundice. This has been the experience of others. Where the distal portion of the duct is unrecognizable, I have employed Walton's method of anastomosing the proximal stump of the duct to the duodenum with variable results. I believe that if an accurate anastomosis of mucosa to mucosa could be carried out the benefit would be more permanent, but there would always be the disadvantage of a direct communication between the duodenum and the bile ducts. It is possible by mobilization of the duodenum and pancreas to demonstrate the distal part of the common duct and to bring it up for anastomosis to the proximal portion without tension.

TABLE II.
Incidence and Mortality in 458 Explorations of Common Duct.

Finding.	Approach.		Total of Operations.	Deaths.	
	Explored through Cystic Duct.	Explored through Common Duct.		Total.	Percentage.
Stones not found	299	27	326	6	1·8
Stones found ..	56	76	132	6	4·5
Total ..	355	103	458	12	2·6

I have made the foregoing remarks to stimulate greater interest in the problem of stricture of the common bile duct and particularly to emphasize the need of early action in bringing about its correction.

Before I conclude, it may be of interest to review the findings of the analysis of 1185 operations performed during the nine years ending December 31, 1947. Table I shows the total death rate (2%); that of cholecystectomy is slightly lower than this figure and that of cholecystectomy with exploration of the common bile duct higher. Table II shows that when stones were removed from the common duct the death rate rose to 4·5%. The tables demonstrate that exploration of the common duct, except when stones have been present, does not add to the risk of the operation, and that there is probably no added risk in the closed method of dealing with a common duct, as compared with drainage. If this is true, as these figures seem to prove, there can be no advantage in external drainage of the common bile duct, provided there is a free passage for the bile to the duodenum. Indeed, the immediate restoration of the unhampered flow of bile to the duodenum must be to the patient's advantage.

LITHIUM SALTS IN THE TREATMENT OF PSYCHOTIC EXCITEMENT.

By JOHN F. J. CADE, M.D.,
Senior Medical Officer, Victorian Department of Mental Hygiene.

LITHIUM SALTS enjoyed their hey-day in the latter half of last century when, commencing with their introduction by Garrod, they were vaunted as curative in gout, and so doubtless in a multitude of other so-called gouty manifestations. This followed the demonstration that lithium urate was the most soluble of the urates. It was shown that if pieces of cartilage with urate deposits were immersed in solutions of sodium, potassium and lithium carbonate, the urate was dissolved first from that piece immersed in the lithium carbonate solution.

As time went on and lithia tablets were consumed on an ever-increasing scale for an ever-increasing range of ailments, the toxic and depressant effects were more and more commonly seen.

Garrod (1859) wrote of lithium carbonate: "When given internally in doses of from one to four grains dissolved in water, two to three times a day, it produces no direct physiological symptom . . . their use does not appear to be attended with any injurious consequences." And certainly, in that dosage, there should never be any toxic symptoms.

But about fifty years later cases are reported "of cardiac depression and even dilatation, as a result of excessive and continued consumption of lithia tablets" (*The Practitioner*, 1907).

"Cardiac depression and even dilatation" was perhaps very vague physiology, but the note of warning was clear, also the statement in Squires's "Companion to the British Pharmacopœia" that "lithia salts upset the stomach very easily" (*The Practitioner*, 1909).

What with the hypothetical cardiac depression and the actual mental depression, nausea and giddiness, the uselessness of lithium in most of the conditions for which it was prescribed, and the fact that there was other, more efficacious treatment in the only disease in which it had been shown to be of some value, it is not surprising that lithium salts have fallen into desuetude.

Culbreth (1927) says of lithium bromide that it is the most hypnotic of all bromides. The dosage stated there is the relatively enormous one of 10 to 30 grains. It is not stated how often this huge dose might be repeated each day, but one presumes the traditional two to three times. Squires, too, states that "in epilepsy it is the best of all bromides" and gives the dose more conservatively as five to 15 grains.

It is worth noting that the hypnotic action of lithium bromide was thought to be due to the fact that, the atomic weight of lithium being so small, weight for weight, lithium bromide must contain more bromide ion than any other bromide. There is no evidence that the lithium ion was recognized as having a marked sedative action superior in some respects to that of the bromide.

But 15 grains of lithium bromide repeated three times a day would soon lead, not to bromide, but to far more dangerous lithium, intoxication, and it is little wonder that it has never found favour in the treatment of epilepsy. It is a pity, because properly used, lithium salts might well be an important addition to the anti-convulsant armamentarium.

In the course of some investigations by the writer into the toxicity of urea when injected intraperitoneally into guinea-pigs, it appeared desirable to ascertain whether uric acid enhanced this toxicity. The great difficulty was the insolubility of uric acid in water, so the most soluble urate was chosen—the lithium salt. When an aqueous solution of 8% urea, saturated with lithium urate, was injected, the toxicity was far less than was expected. It looked as if the lithium ion might have been exerting a protective effect. To determine this, more observations were made, lithium carbonate being used instead of lithium urate. An 8% aqueous solution of urea kills five out of ten guinea-pigs when injected intraperitoneally in doses of 1·25 millilitres per ounce of body weight. When 0·5% lithium carbonate in an 8% urea solution was injected in the same dosage, all ten animals survived; and this argued a strong protective function for the lithium ion against the convulsant mode of death caused by toxic doses of urea.

To determine whether lithium salts *per se* had any discernible effects on guinea-pigs, animals were injected intraperitoneally with large doses of 0·5% aqueous solution of lithium carbonate. A noteworthy result was that after a latent period of about two hours the animals, although fully conscious, became extremely lethargic and unresponsive to stimuli for one to two hours before once again becoming normally active and timid.

It may seem a long distance from lethargy in guinea-pigs to the excitement of psychotics, but as these investigations had commenced in an attempt to demonstrate some possibly excreted toxin in the urine of manic patients, the association of ideas is explicable.

It appeared worth while in view of these results to try lithium salts in the treatment of two distinct disorders —firstly mania, in view of their sedative effect; secondly epilepsy, in view of their anti-convulsant action. With the latter, this paper is not concerned.

Henderson and Gillespie (1944) remark, in their historical survey of psychiatry, that the waters of certain wells were considered to have special virtue in the treatment of mental illness, and mention some of the more famous in the British Isles. It is very likely that their supposed efficacy was a real efficacy and directly proportional to the lithium content of the waters.

In the treatment of such a self-limiting disorder as mania, the therapeutic innovator must be more than ordinarily on his guard. Whether this or that treatment is of any value must be carefully assessed from as many angles as possible. With an episodic disorder of this type, the efficacy of a particular treatment may be judged by one or more of the following criteria. The more criteria that are satisfied, the more sure are we that it is a treatment of real and not suppositious value.

1. Improvement must proceed *pari passu* with treatment.

2. Chronic cases are of special value in assessment because in them spontaneous remission is far less likely to occur in a specified short period than in the recent cases.

3. Supporting evidence may be forthcoming from the treatment of non-manic psychotic excitement.

4. The ideal is, of course, the method of controlled observation of a sufficient number of treated and untreated patients. The disadvantage of the ideal is that mania is not so common a psychotic disorder as might be thought and it would take any one observer even in a large mental hospital probably some years to accumulate a large enough series to be statistically significant. But although the first three criteria may be insufficient for formal proof, they are capable of giving strong circumstantial evidence for or against efficacy.

So far ten manic patients have been treated, of whom three suffered from chronic and the remainder from recurrent mania. In addition, similar treatment was given to six patients with *dementia præcox* and three melancholics.

Case Histories.

CASE I.—W.B., a male, aged fifty-one years, who had been in a state of chronic manic excitement for five years, restless, dirty, destructive, mischievous and interfering, had long been regarded as the most troublesome patient in the ward. His response was highly gratifying. From the start of treatment on March 29, 1948, with lithium citrate he steadily settled down and in three weeks was enjoying the unaccustomed surroundings of the convalescent ward. As he had been ill so long and confined to a "chronic ward", he found normal surroundings and liberty of movement strange at first. Owing to this, as well as to housing difficulties and the necessity of determining a satisfactory maintenance dose, he was kept under observation for two months. He remained perfectly well and left hospital on July 9, 1948, on indefinite leave with instructions to take a maintenance dose of lithium carbonate, five grains twice a day. The carbonate had been substituted for the citrate as he had become intolerant of the latter, complaining of severe nausea. He was soon back working happily at his old job. However, he became more lackadaisical about his medicine and finally ceased taking it. His relatives reported that he had not had any for at least six weeks prior to readmission on January 30, 1949, and was becoming steadily more irritable and erratic. He ceasedl work just before Christmas. On readmission to hospital he was at once started on lithium carbonate, ten grains three times a day, and in a fortnight had again settled down to normal. The dose of carbonate was then reduced to five grains three times a day, and in a further two weeks to five grains twice a day. He is now (February 28, 1949) ready to return to home and work.

CASE II.—E.A., a male, aged forty-six years, had been in a chronic manic state for five years. He commenced taking lithium citrate, 20 grains three times a day, on May 5, 1948. In a fortnight he had settled down, was transferred to the convalescent ward in another week, and a month later, having continued well, was permitted to go on indefinite trial leave whilst taking lithium citrate 10 grains three times a day. This was reduced in one month to 10 grains twice a day, and two months later to 10 grains once a day. Seen on February 13, 1949, he remained well and had been in full employment for three months.

CASE III.—P.B., a male, aged forty years, was suffering from a second attack of mania which had already lasted five months and showed no signs of clearing up. He commenced lithium citrate, 20 grains three times a day, on April 5, 1948, and in a week was sufficiently settled down to go to the convalescent ward. He continued to improve over several weeks and remained well. On January 31, 1949, he reported for follow-up. He continued well and had been in full employment for over three months. He had been on a maintenance dose of five grains of lithium carbonate twice daily. He was advised to continue with five grains once a day.

CASE IV.—T.F., a male, aged sixty-three years, was suffering from chronic mania of two and a quarter years' duration. There was a strong history of alcoholism with some evidence of senile enfeeblement. He was continuously garrulous, restless, irritable and euphoric. He commenced taking lithium citrate, 20 grains three times a day, on June 15, 1948. He forthwith began to quieten down and although citrate had to be discontinued on June 28, 1948, because of nausea and malaise, by June 30, 1948, he was quiet and capable of rational conversation. His euphoria and excitement had quite disappeared only to leave a rather irritable and credulous old man. He recommenced taking lithium citrate, 10 grains three times a day, but in view of constant abdominal discomfort the carbonate, five grains three times a day, was substituted on July 8, 1948. As his discomfort persisted and even when no longer manic he was still a mildly enfeebled, irritable old man, it hardly seemed worth while to persist and treatment was discontinued on August 10, 1948. In a fortnight he had drifted back to his previous manic state.

CASE V.—B.D., a male, aged forty-four years, was suffering from a recurrent manic episode. The present attack had lasted two and a half months and showed no signs of abating. He was restless, noisy, elated, with marked distractibility and flight of ideas. He commenced taking lithium citrate, 20 grains three times a day, on July 30, 1948. By the end of the first week he had shown some improvement. This continued steadily for a further two weeks, by which time he was quite normal. On August 27, 1948, the dose of citrate was reduced to 10 grains three

times a day for one week, with instructions to the patient, an intelligent man, who was then leaving hospital, to take 10 grains twice a day for a further week and then to continue on 10 grains at night indefinitely. He has remained well.

CASE VI.—A.M., a man of sixty years, suffered from manic-depressive insanity associated with alcoholism. His previous attacks had been mainly depressive, but he had had a manic phase lasting five months two years previously. By November 17, 1948, he had been developing a manic phase for a fortnight, steadily worsening until now he was noisy, restless and aggressive. On this date he commenced taking lithium citrate 20 grains three times a day. In a week he was settling down, but at the end of a fortnight the administration of lithium citrate had to be temporarily discontinued as he was showing toxic symptoms—he was asthenic and tremulous, with slurring speech. The toxic symptoms disappeared in four days and citrate administration was resumed with a dose of 10 grains three times a day. By this time he had settled down completely. On February 14, 1949, after lithium citrate administration had been discontinued for seven weeks, he was again becoming unsettled and losing weight. Given lithium citrate 20 grains three times a day, he once again settled down promptly in four days, and at the end of a week when he had put on three pounds in weight the dose was reduced to a maintenance dose of 10 grains once daily.

CASE VII.—M.C., aged forty years, was suffering from recurrent mania. In this episode he had been excited, restless and violent for over two months and was so interfering that he often had to be confined to a single room during the day. On February 7, 1949, he commenced taking lithium citrate 20 grains three times a day. In four days he was distinctly quieter and by February 13, 1949, appeared practically normal. He continued well and on February 20, 1949, the dose of citrate was reduced to 10 grains three times a day. He left hospital on February 27, 1949, with instructions to take 10 grains three times a day for a further week, 10 grains twice a day for a further two weeks, and then 10 grains at night indefinitely.

CASE VIII.—W.M., a man of fifty years, was suffering from an attack of recurrent mania, the first of which he had had at the age of twenty. The present attack had lasted two months and showed no signs of abating. He was garrulous, euphoric, restless and unkempt when he started taking lithium citrate twenty grains three times a day on February 11, 1949. Two days later he was reported to be quieter. By the ninth day he was definitely settling down and the following day commenced work in the garden. By the end of two weeks he was practically normal—quiet, tidy, rational, with insight into his previous condition. This was in marked contrast to his condition a fortnight before when he had to be locked in a single room at night with a regular nocturnal hypnotic and was too restless to eat in the dining room owing to his unsettling effect on the other patients.

CASE IX.—W.S., a powerfully built man of forty-seven years, had suffered from recurrent manic phases since the age of twenty-five years. He last left hospital after a stay of seven months on August 31, 1948, whilst still in a hypomanic state and appears to have remained thus until his condition became worse and he was readmitted to hospital on February 11, 1949, in a state of typical manic excitement. Fortunately, in view of his physique, he is good humoured and never becomes violent. On February 11, 1949, he commenced taking lithium citrate three times a day. He was considerably quieter two days later, was working happily in the kitchen in a few days, and by the ninth day was practically normal. On February 27, 1949, as he was remaining well, the dose of citrate was reduced to 10 grains three times a day. On March 1, 1949, he was complaining of mild malaise and abdominal discomfort and administration of the drug was discontinued for a few days. He recommenced taking lithium citrate 10 grains twice a day on March 4, 1949. An acquaintance who has known the patient for years reports that he has never seen him as normal as at present.

CASE X.—R.T., a man of sixty-one years, presents several points of interest. He has had manic episodes for twenty-eight years, the attacks lasting from three to ten months. He was readmitted to hospital on January 5, 1949, in his usual noisy, elated, restless state with depraved habits. He is so excited in such phases that it is impossible to determine whether or not he is hallucinated or delusional. He commenced taking lithium citrate 20 grains three times a day on January 28, 1949. On February 3, 1949, he was quieter, but mildly toxic—dizzy, unsteady and nauseated. Lithium citrate administration was discontinued, and on February 7, 1949, when the toxic symptoms had disappeared and the patient was becoming grandiose and truculent again, he was started on lithium carbonate 10 grains three times a day. By February 19, 1949, it was evident that his excitement was abating steadily, but it was also becoming obvious that he was also constantly hallucinated and delusional, muttering to himself as he communicated "by telepathy" with various people. This state continued, that is, an excited delusional state in which the excitement was well controlled by lithium, but the delusional state was quite unaffected. Whether such a case can be regarded as one of true mania is a matter upon which there may well be considerable difference of opinion.

In addition to these ten patients, six patients suffering from *dementia præcox* were treated with lithium citrate, 20 grains three times a day, for from three to four weeks. An important feature was that, although there was no fundamental improvement in any of them, three who were usually restless, noisy and shouting nonsensical abuse, paralleling the patient in Case X, lost their excitement and restlessness and became quiet and amenable for the first time for years. The taking of a nocturnal hypnotic had been a routine and could be discontinued during treatment. They reverted to their previous state upon cessation of lithium medication.

It would be natural to suppose that as lithium salts cause the symptoms of mania to subside, continued dosage might precipitate a depressive episode in predisposed persons. So far there is no evidence of this. Three patients suffering from chronic depressive psychoses were given, for several weeks, lithium citrate in the same dosage as that prescribed for mania patients. There was no improvement, but neither was there any aggravation of the depression.

Dosage, Over-Dosage, Maintenance Dose.

The British Pharmacopœia gives the dose of lithium carbonate as two to five grains and that of lithium citrate as five to ten grains, but such figures convey little information of value in therapeutics in the absence of any information as to how often such a dose may be given in each twenty-four hours, or of the rate of elimination.

Culbreth (1927) is more liberal and gives the dose of lithium carbonate as five to 15 grains and of the citrate as 10 to 30 grains.

In practice one finds that some patients can tolerate lithium citrate 20 grains three times a day for weeks without toxic symptoms, but that a high proportion show toxic symptoms in one to three weeks on such a dose.

It seems advisable to keep the patient on a maximum dose—that is, lithium citrate 20 grains three times a day or lithium carbonate 10 grains three times a day—whilst he continues to improve. Once normal emotional tone is attained the dose is progressively reduced: lithium citrate to 10 grains three times a day for one to two weeks, to 10 grains twice a day for a further one to two weeks, and then a maintenance dose of 10 grains after the evening meal indefinitely. The corresponding doses of lithium carbonate are half those for the citrate. In view of their liability to produce gastric upsets lithium salts are given after meals.

The reason for using two alternative preparations is that the citrate is very soluble and appears to be better absorbed than the carbonate, whereas the carbonate must be put up suspended in mucilage or given in capsules. However, the carbonate has the advantage that it is better tolerated by some patients and appears less liable to produce either gastric disturbances or other toxic symptoms.

The symptoms of over-dosage are referable mainly to the alimentary and nervous systems. Abdominal pain, anorexia, nausea and vomiting occur and occasionally mild diarrhœa. The nervous symptoms are giddiness, tremor, ataxia, slurring speech, myoclonic twitching, asthenia and depression. The patient looks ill—pinched, drawn, grey and cold.

Unless such symptoms are followed by immediate cessation of intake there is little doubt that they can progress to a fatal issue. It is therefore of the utmost importance that when a patient is on maximum doses he should be seen each day and that the nursing staff

should be instructed to look for early symptoms of saturation.

If toxic symptoms develop, they disappear quickly—that is, in two to four days—when the drug is completely withdrawn. Treatment may then be resumed with a smaller dose, or, if it is still desirable to use a maximum dose, by substituting the carbonate for the citrate.

Discussion.

There is no doubt that in mania patients' improvement has closely paralleled treatment and that this criterion has been fulfilled in the chronic and subacute cases just as closely as in the cases of more recent onset. The quietening effect on restless non-manic psychotics is additional strong evidence of the efficacy of lithium salts, especially as such restlessness returned on cessation of treatment.

Lithium salts have no apparent hypnotic effect; the result is purely sedative. The effect on patients with pure psychotic excitement—that is, true manic attacks—is so specific that it inevitably leads to speculation as to the possible ætiological significance of a deficiency in the body of lithium ions in the genesis of this disorder.

Lithium may well be an essential trace element. It is widely distributed, has been detected in sea-water and in many spring and river waters, in the ash of many plants, and in animal ash.

Pre-frontal leucotomy has been performed lately on restless and psychopathic mental defectives (Mackay, 1948; Engler, 1948) in an attempt to control their restless impulses and ungovernable tempers. It is likely that lithium medication would be effective in such cases and would be much preferred to leucotomy.

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SKIN LESIONS AMONG FISHERMEN AT HOUTMAN'S ABROLHOS, WESTERN AUSTRALIA, WITH AN ACCOUNT OF ERYSIPELOID OF ROSENBACH.

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Introduction.

IN the course of an investigation throughout 1947 into the biology of the marine crayfish, *Panulirus longipes*, at Houtman's Albrolhos, a group of coral islands situated about forty miles off the Western Australian coast at Geraldton, it was evident that the efficiency of the fishermen and crayfish processors generally, and of the research workers, was impaired by the presence of severe skin lesions primarily on the hands, knees and feet and secondarily on the nape of the neck and around the anal region. Accordingly a side investigation was carried out into the causes, prevention and methods of treatment of the lesions, forty-three fishermen and other workers being examined. The first named author was responsible for the field-work, treatment, and the limited bacteriological tests at the islands. The second author, who was unable to visit the area, acted as consultant during the investigation and the preparation of this report.

Working Conditions.

The islands occupied by the fishermen during the fishing season (March to August) are low-lying areas of coral shingle and old coral limestone surrounded by coral reefs. Crayfish are caught in cray-pots, are stored in slotted crates and, when required, are bagged for transport to Geraldton. There is thus much handling of these spiny animals. Much of the bait that is used is brought from the mainland and consists of cow hocks, sheep heads, and pieces of horse flesh, all in varying stages of decomposition; the balance consists of reef fish whose spines are capable of inflicting deep puncture wounds. Scratches, cuts and punctures are common and invariably become infected under ordinary working conditions. Flies are plentiful. The hands and legs are immersed constantly in salt water, while fresh water, which is brought from Geraldton, is not provided in adequate quantity. Food is plentiful and in some cases well varied; but, in general, there is a heavy stress on spaghetti, white bread, potatoes and fish. Geraldton is the nearest centre for medical attention, but the fishermen rarely seek this unless they are in advanced or crippling stages of illness. It was therefore required to institute curative and preventive measures on the spot, and necessary to examine what primary conditions and infections existed.

The fishermen realized that underlying factors were present and to these, in general, they gave the name "crayfish" or "coral" poisoning. This aspect was examined. No evidence was obtained of the presence of toxic substances directly associated with the crayfish, but the nematocysts of the live corals caused slight irritations which quickly faded, and beyond being responsible, through transference to the eyes, for mild conjunctivitis, caused little trouble.

Generalized Clinical Picture.

The commonly occurring lesions could be grouped into five general types. Often all occurred in the same case at the same time.

1. The first type comprised those which developed from small cuts and abrasions on exposed areas as small, angry, red macules, with local throbbing and irritation followed by considerable pus, necrosis of the overlying tissues, and the formation of characteristically shallow ulcers. Occasionally, lymphangitis developed, but rarely were either the inguinal or axillary glands involved, the temperature did not rise greatly (0·5° to 1·0° F.) and the pulse rate was but slightly altered. Streptococci predominated in cultures from these sites.

2. The second type of lesion developed as shallow or deep swellings at the site of either deep or shallow injuries which tended to heal rapidly at the surface, leaving a deepening, pus-filled cavity below, the healed surface breaking down at a later date to form a deep ulcer. With puncture wounds, which had not bled freely, infections of this type were very frequent. Staphylococci predominated in cultures from these sites.

3. A third type, appearing as a rule toward the end of the fishing season, were lesions which developed as isolated centres of infection and which varied from small vesicles to furuncles and abscesses with no history of cuts or abrasions. The general sites of this type of infection were the upper part of the forearm, from the knee to the buttocks, and the nape of the neck, although any part of the body could be involved. Staphylococci predominated in cultures from these lesions.

In many cases in which small furuncles were formed the entry was apparently through hair roots and skin glands. The areas, if neglected, became the site of spreading ulcers through streptococcal reinfection. In other cases in which deep-seated abscesses formed, the

source was most probably systemic and indicative of a high degree of mass infection.

4. Ulcers termed by the fishermen "salt water sores" were a distinct entity. They were characteristically rounded or oval in outline and were grouped; the margins were not greatly raised above the general level of the skin and the whole of the floor of the ulcer was more or less evenly depressed to the level of the subcutaneous layers. Healing was very slow, tending to take place from the circumference rather than from the floor of the ulcer, which usually obtained its maximum size within several days and persisted at this for weeks and months. It was apparent that they originated in necrosed patches which became the site of a mass infection, the probable cause being the constant immersion of the part in salt water, thus devitalizing areas of skin in a subject already predisposed to this condition through nutritional deficiencies. It was noticeable that the greatest incidence of this type of ulcer was among those whose starch intake, either in the form of spaghetti, peeled potatoes or white bread, was the highest.

5. Two closely allied types of infection became apparent on further examination. The first were surface lesions, apparently similar to those described in the first type but spreading rapidly in the surface skin layers instead of remaining more or less static. In addition a degree of stiffness of adjacent joints occurred with some pain. There was general weakness, with headache, loss of weight, irritability and restlessness. These major symptoms also characterized a second form in which deep-seated swellings followed on puncture wounds, and which simulated a pyogenic infection without, however, any pus formation. After the healing of the entrance wound a hard area remained at a depth with loss of sensitivity in the surface layers and deep-seated pain on pressure. With both types the immediately affected area, together with adjacent areas, was often swollen and painful with the skin hard and shiny. There was a rise of temperature (1·0° to 2·0° F.), the pulse rate increased and often became irregular, while adjacent lymph glands became hardened. The acute period was passed through fairly rapidly (five to ten days), but subsequent recovery was slow (three to five weeks) with often subacute relapses after that time.

Closer examination of the spreading surface lesions showed that, preceding the pus-filled ulcer, there occurred a narrow margin which was purplish-red, showed no evidence of pus formation, and advanced fairly quickly, the area behind rapidly breaking down. The presence of the advancing erythema suggested that some hitherto unrecognized non-pyogenic organism was present. On the basis of the similar general symptoms it was felt that the same organism was initially responsible for the both of these infections of the fifth class. The surface erythema uncomplicated by a secondary infection developed in one of us (K.S.) and the following study was made.

A purplish-red erythema developed at the site of a small abrasion on the outer side of the first phalanx of the first digit of the left hand and steadily spread toward the base of the finger, across the web, and up along the second finger. The affected area was swollen and tight and presented a fair degree of burning and itching. The first and second joints of the two fingers and the wrist joints were stiffened and painful. There was a slight temperature rise (1·0° F.) with headache and lassitude. Lymphangitis was not present.

The affected area on the first finger was cleansed and protected from secondary infection with a sterile dressing; there was no suppuration and no desquamation beyond a very slight scaling. When the erythema had spread to the second finger, the protection was removed from the first finger and that area was left exposed to the ordinary risks of infection during the normal work. Rapid over-all disintegration of the skin of the exposed area followed and a typical ulcer of the fifth type ensued. This was treated, healing satisfactorily.

Attempts were made under field conditions to recover organisms by culture from the spreading margin of the erythema, but only the usual flora was encountered until sections were taken from the corium at that point. Predominant among the organisms present in those cultures were slim, very slightly bent rods (length 1·0 to 1·5 μ) in chains, non-motile, non-spore-forming, and Gram-positive (Gram-Weigert stain). Growth was good on plain agar, provided the sections, or scrapings from them, were immersed therein. It was scanty on blood agar and there was no growth on potato. In the field it was difficult to keep incubation temperatures steady and these varied between 30° C. and 40° C. There was no liquefaction in gelatine stab cultures and the growth was of the brush type. Since the infection appeared to be sulphonamide-resistant a trace of soluble thiazamide was incorporated in the agar plates. Growth of the organism was good and it was possible to obtain pure streak cultures. These were subcultured at a higher temperature (40° to 45° C.) in order to obtain some degree of attenuation and an inoculation was made on the outer side of the wrist of one of us (K.S.). After three days a sharply defined erythema developed which spread chiefly in one dimension from the point of inoculation. When the growing point on one side was half an inch from the inoculation point a section of the corium there was cultured and the organism recorded in quantity.

There appeared no doubt but that the described organism was the one responsible for the spreading erythema, while a search of the literature showed that the clinical picture closely accorded with that of erysipeloid of Rosenbach in a relatively mild form. As the work was done in the field under considerable difficulties the bacteriological tests were incomplete and no serological tests were made; however, the facts that were obtained were not inconsistent with a determination that the causative organism was *Erysipelothrix rhusiopathiæ*. The only object of the bacteriological work was to assist in developing curative methods; treatments based on the above findings proved satisfactory.

Erysipeloid of Rosenbach.

Four clinical types of infection of swine by *Erysipelothrix rhusiopathiæ* are recognized, namely, urticarial ("diamond back"), arthritic, acute or septicæmic, and chronic or cardiac. It has also been shown that the domestic fowl, pigeons, turkeys, sheep, rats, and possibly the horse are subject to infection by this bacillus. In man infection is followed by a variety of lesions and clinical states, ranging from a mild erysipeloid to polyarthritis and fatal endocarditis; it occurs chiefly among workers in the fish, crab and crayfish industries, among caterers, and among abattoir and stockyard employees. In Western Australia it has been recognized only in fish and crayfish workers, among whom it occurs in forms ranging from a mild erysipeloid to a mild polyarthritis, suggesting that the organism responsible is present in an attenuated form. The subject is well covered by the following references: Russell and Lamb (1940), Klauder *et alii* (1943), Klauder and Rule (1944), Schwartz and Tabershaw (1945), Barber *et alii* (1946), King (1946), Klauder (1947), and a general report in the *British Medical Journal* (1948).

> The causative organism is widely disseminated, its habitat being dead matter of plant or animal origin. In certain environments it exists in the soil as a saprophyte or as a virulent pathogen. These qualities are not stable since *E. rhusiopathiæ* may be alternately virulent or avirulent. The organism has been recovered from the slime of fish, from house-flies and from putrefying horse-flesh. Klauder (1947, page 135).
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> While the organism is destroyed quickly by boiling water and, within a few minutes, by the usual disinfectants, it will remain alive in the dark for a month or more. In salted or pickled meats it will remain alive for 3 or 4 months and in putrid material it is capable of retaining its viability and virulence for months. Klauder *et alii* (1947, page 135).

Writers in general stress the need for recognizing that the infection may be very widespread and relatively common. For instance, King (1946) records 115 cases out of 2300 at the Septic Hand Clinic at Charing Cross

from 1941 to 1945. It is suggested also that *Erysipelothrix rhusiopathiæ* septicæmia should be considered when the clinical diagnosis is subacute bacterial endocarditis with a blood culture report of a Gram-positive rod, providing the organism is non-motile and non-spore-forming. This particularly is so when there is an increase in the monocytes (Klauder *et alii*, 1943).

In view of the known incidence of infection of sheep by the bacillus in the eastern States of Australia—for example, polyarthritis of lambs, and cutaneous infections following dipping in contaminated dipping fluids—attention should be paid to the possibility of *Erysipelothrix rhusiopathiæ* in skin lesions, polyarthritis, and endocarditis occurring among workers there in the meat and allied industries, and, because sheep heads are used as bait, among crayfish fishermen.

Conclusions.

The over-all clinical picture indicated that the ulcers arose in three ways: firstly, by the entry of organisms, chiefly *Staphylococcus*, *Streptococcus* and *Erysipelothrix*, through cuts, abrasions and punctures; secondly, by the entry of staphylococci and streptococci through areas of devitalized and necrosed skin; and thirdly, by the entry of staphylococci through hair roots and skin glands. Methods of prevention and treatment were investigated on this basis during 1947 with the result that the incidence of lesions was greatly reduced during 1948, while those which occurred were readily controlled.

Prevention.

The measures adopted included: the provision of protective gloves, either of rubber or of canvas, long-gauntleted with the thumb separate and the four fingers in one; greater care in the handling of crayfish, pots and bait; the prompt cleansing of infective organisms from wounds and immediate treatment with antiseptic agents together with greater attention to camp and boat hygiene; and care in washing the hands and legs with fresh water after exposure to salt water, this being followed by thorough drying and the application of boric acid talc where the skin was soft and soggy. Finally, attempts were made to provide an adequately varied diet including fresh fruit and vegetables, while fresh water was transported to the islands in greater quantity.

Treatment.

Treatments were not easy to apply in simple cases and in preliminary stages, as the fishermen and operatives carried on with their work so that dressings speedily became soaked, soiled and useless. Adhesive tabs, frequently changed, were useful on sites not exposed to water, but otherwise they increased the possibility of the occurrence of devitalized skin areas with consequent increased ulceration. After some experiment it was found that early infections uncomplicated by salt water sores were controlled well by washing the site with a standard antiseptic such as "Dettol", "Ellisol", hydrogen peroxide or potassium permanganate, and by painting the area with "Metaphen" tincture (4-nitro-anhydro-hydroxy-mercury-ortho-cresyl, Abbott); failing this, flavine or gentian violet was of use. Ichthammol was not particularly effective unless the area could be protected. Neglected and more severe infections required the repeated use of "Metaphen" packs, or, more effectively, of either "Monacrin" (5-amino-acridine-hydrochloride), or ichthammol in 12% alcohol, packs changed two-hourly.

When erysipeloid was present as a mild infection either on the extremities or on the anal region, painting with "Metaphen" tincture or the use of wet packs of "Monacrin" was effective. If the infection was extensive or deep-seated, wet packs of "Monacrin" or dressings of ichthammol in 12% alcohol were usually sufficient if coupled with splinting and immobilization of the affected part. In more severe cases, with the appearance of arthritic and systemic symptoms, penicillin at the rate of 15,000 to 20,000 units every three hours for twenty-four hours appeared to be sufficient. Sulphonamides are useless for this purpose; see also Klauder and Rule (1945) and Barber *et alii* (1946). Specific serum has been tested in some cases overseas; but serum sickness was very common and was very often more trying than the infection itself (report, *British Medical Journal*, 1948).

"Salt water sores" in uncomplicated cases are very like those of the deficiency dermatitis "Barcoo rot" without heavy scabbing, the sores somewhat resembling a characteristic impetigo. They are often masked by lesions resulting from the infection of accidental injuries, but may be recognized by their clear-cut circular or oval shape and by their grouping in clusters. At the suggestion of one of us (H.G.D.), an empirical treatment similar to one used at one time against "Barcoo rot" was tested. One tablet (five grains) stannic oxide was administered after each meal until a reverse effect was obtained, the margins of the ulcers commencing to ring with red. Administration was then stopped, the accompanying fresh water washes followed by antiseptic painting being continued. The treatment was satisfactory, the ulcers clearing up within two to three days on one of us (K.S.), and on eight fishermen who were chronic sufferers.

Summary.

Examination of 43 fishermen at the Abrolhos Islands, Western Australia, during 1947 showed the presence of many lesions resulting from the infection of superficial wounds by staphylococci, streptococci, and erysipelothrix, among other agents. The incidence on the extremities of devitalized skin areas on which ulcers ("salt water sores") formed was high. Preventive measures introduced included protection of the hands with gloves, greater attention to personal and general cleanliness, and the use of a more varied fresh diet.

Treatments included the use of "Metaphen", "Monacrin" and ichthammol as dressings; stannic oxide, given orally, against salt-water sores; and penicillin therapy in more serious cases when lymphangitis was present. Penicillin was the treatment of choice in cases involving more serious forms of erysipeloid, as these were resistant to the sulphonamides.

It is suggested that erysipeloid of Rosenbach, which may be masked by secondary infections that are difficult to control until the primary erysipeloid is treated, may prove to be widespread among fishing and fish-processing communities in Australia. Overseas experience has shown that this disease is relatively common, ranging from mild erysipeloidal skin lesions to septicæmia and endocarditis.

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PNEUMONOKONIOSIS ON WESTERN AUSTRALIAN GOLDFIELDS.[1]

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THE commencement of gold mining in Western Australia occurred about 1885, when gold was discovered at Hall's Creek in the Kimberleys. Within a few years thereafter mining centres had been established in the Pilbarra district (Marble Bar), the East Murchison (Mount Magnet, Meekatharra, later Wiluna), the Yilgarn (Southern Cross) and at Coolgardie, Kalgoorlie, Leonora and Norseman.

The centre of the industry was eventually established at Kalgoorlie, where the area known as the "Golden Mile" became world famed for its rich ore deposits.

For several decades the number of men employed in the industry has fluctuated between 5000 and 15,000, from 50% to 60% being employed on the "Golden Mile".

At the beginning of the century the problem of pneumonokoniosis assumed major proportions throughout the industry. In 1911, Dr. J. H. L. Cumpston, appointed a Royal Commissioner to inquire into the prevalence of pulmonary diseases amongst miners, found evidence of fibrosis of varying degree in 22% of 1805 working miners examined, and of active tuberculosis in 1·5%.

In 1925 to 1926 the Commonwealth Health Laboratory, Kalgoorlie, at the request of the Western Australian Government, conducted a clinical and radiological survey of 3039 men employed on the "Golden Mile" and 1028 men employed in the industry in outlying centres. Of the total, 642 (or 16%) were found to be suffering from silicosis, 131 (or 3·3%) from silicosis with tuberculosis, and 11 (or 0·3%) from tuberculosis only.

In the years that have followed this initial survey the laboratory has continued to carry out certain functions on behalf of the Western Australian Government and the mining industry. These are, first, the pre-employment examination of all entrants to the industry, and the exclusion from underground employment of those considered "poor risk" subjects from the industrial disease standpoint. Since the completion of the initial survey, some 34,000 individuals have undergone the pre-employment examination. The rejection rate has varied from 5% to 15%, indications for rejection comprising, in the main, evidence of past or present pulmonary disease, disease of the circulatory system, the effects of old injuries, dermatitis, hernia and spinal rigidity.

In addition to these pre-employment examinations, the laboratory has been responsible for carrying out periodic reexamination of working miners, underground men being examined at yearly intervals, and most of the others at least every two years.

The results of these examinations show that, in the twenty-one years 1927 to 1947, a yearly average of 5500 reexaminations have been performed. In the same period a further 1162 men have developed silicosis (an average annual incidence of 1% of the men examined). There have also been found 169 further cases of tuberculosis in men presenting no signs of pneumonokoniosis, and 539 cases of tuberculosis with pneumonokoniosis, that is, tuberculosis superimposed on any radiological stage of fibrosis from accentuated reticular markings to advanced nodular or massive consolidation.

The total number of cases of tuberculosis, with or without accompanying pneumonokoniosis, diagnosed from the commencement of the initial survey, in September, 1925, to the end of 1947, was 839. In 538 of these cases the men concerned were excluded from the industry before 1931, and the average annual incidence, with very narrow fluctuations, for the fifteen years 1933 to 1947, was 13, or one per 400 men examined.

Follow-up reexaminations of silicotic ex-miners have been carried out on an increasing scale in the past ten years, and the results point to a considerable reduction in the total incidence of tuberculosis among miners and ex-miners, such incidence being estimated to be, at the present time, approximately 20 per year. Under present conditions the silicotic miner who is destined to become tuberculous will not usually do so during his working career, but as a terminal event, late in life, many years after the cessation of exposure to silica dust.

[1] Read at a meeting of the Section of Radiology and Radiotherapy, Australasian Medical Congress (British Medical Association), Sixth Session, Perth, August, 1948.

The incidence of simple silicosis, although somewhat inflated during the war years as a result of the enforced readmission of old partly dusted miners to the industry, is now returning to the level of the immediate pre-war years, when new cases appeared at the rate of 20 to 25 per year. This may be compared with an average incidence of 70 per year for the eight years 1929 to 1936.

In general, it may be said that the introduction, about 1923, of preventive methods, which were at that time considered adequate by South African standards, and the attention paid to ventilation since that time, have succeeded in reducing by two-thirds the silicosis and tuberculosis production rates, and in increasing, by one-half at least, the average length of time required for the development of silicosis in underground mine workers.

There are at present about 200 men with classical nodular silicosis remaining in employment and scattered throughout the industry. The great majority of these men have been transferred to surface work within their capacity. Of the other 1600 cases diagnosed since the commencement, those men who have become tuberculous have been compulsorily retired, and the remainder have voluntarily left the industry, on partial or total compensation, usually within a few years of their first notification.

The above figures relate mainly to silicosis and tuberculosis. Other types of pneumonokoniosis are rarely encountered as disabling conditions amongst employees in the metalliferous mines. Pre-silicotic changes, however, are very common in gold miners, particularly in underground miners who have been employed for ten years or more in the industry. On the "Golden Mile", for instance, one in every three underground men examined in 1948 had radiological evidence of early fibrosis, usually of reticular pattern.

These pre-silicotic changes are generally referred to by the laboratory under the heading of "increased fibrosis". Their significance will be discussed later.

Procedure.

Limitations of staff preclude the carrying out of clinical examinations of such large numbers, and in practice the annual reexamination of a mine worker is primarily by X-ray examination of the chest only. One in every eight or ten men, those whose films show abnormalities considered significant, are subjected to clinical examination and such other follow-up as can be managed with the facilities available. Where necessary they are referred to the tuberculosis authorities for more complete investigation. It is customary at each reexamination to record details of the industrial history since the preceding examination.

The pre-employment examination includes the recording of the previous industrial history, full clinical examination, X-ray examination of the chest and the carrying out, where necessary, of ancillary investigations.

At Kalgoorlie a modern high-power X-ray plant and the facilities of a pathological laboratory are available. Outlying districts are covered by a transportable 30-milliampere X-ray machine, with accessories, and the "travelling laboratory" is staffed by a medical officer and a radiographer. In outlying centres it is customary, and usually necessary, to conduct the examinations at the individual mines. Naturally, the radiographs obtained from the travelling unit do not always compare with those obtainable with the rotating anode tube, but with the employment of short-distance technique and fast screens, a surprisingly good and uniform standard is obtained.

For accurate diagnosis and grading of industrial fibrosis, good radiographs are, of course, essential. As in other similar departments, it is found that variations in density and clarity of the films, leading to difficulties in interpretation, are more often the result of some failure of technique in the processing room than in the X-ray room. This applies to both the Kalgoorlie laboratory and the travelling unit. Under the conditions of use, especially on the travelling unit, the effective life of intensifying screens is short, and "unsharpness" and other faults arising from this factor cause ever-recurring problems; so much so that experiments with "non-screen" techniques have been made.

All films and records are stored permanently at the Kalgoorlie laboratory.

Radiological Abnormalities.

Abnormal findings in pneumonokoniosis as it occurs in Western Australia, so far as the lung parenchyma is concerned, may be resolved into two basic components, accentuation of

the reticular markings and nodulation. Each has a typical pathological counterpart. Massive shadows are nowadays rarely seen in the films of working miners and because of limitation of space will not be further considered.

Reticular Accentuation.

Reticular markings in the radiograph are not quite so specific as nodulation, but where in a working miner or ex-miner from Western Australian goldfields whose occupational dust hazard is known, reticular accentuation is diffusely and symmetrically present in both lung fields, and more or less uniform from apex to base and from the immediate prehilar zone to the periphery, being very well seen towards the costo-phrenic angles, unless there is other obvious cause industrial fibrosis may be confidently diagnosed. Occasionally the condition is developed earlier and is most marked in the middle zones of the lungs. It may be "soft" in appearance, and easily masked by over-penetration, or "hard" and not so easily masked, the latter appearance signifying a firmly established fibrosis. In some cases, on close inspection, fine mottling at the intersections of the linear network can be discerned. However, the essential feature, the outstanding impression given by the film, is that of a diffuse, even network.

In a good radiograph it is not difficult to distinguish a typical reticular accentuation from the branching vascular shadows of arteriosclerosis or other non-industrial disease processes affecting blood vessels. In pneumonokoniosis, as in certain other diseases, the seat of the pathological process is in the lymphatic system. The full-blooded lungs of some adolescents give an X-ray picture which is as a rule comparatively easily distinguished. A mild degree of reticular accentuation, usually of uneven confused pattern, is not uncommonly seen in stokers, firemen, boiler cleaners, and men who have been employed for some years in the timber milling industry. In films which are technically imperfect, difficulty may be experienced in defining the borderline of normal.

The condition (or stage) of reticular accentuation appears in the X-ray films of Western Australian gold miners as a precursor of silicotic nodulation, just as in many coal miners it precedes the development of the condition known radiologically as "reticulation", the latter actually being a fine, closely packed nodulation. Occasionally, when the onset of nodular silicosis is rapid, there is no discernible intermediate stage of reticular accentuation.

Nodulation.

As with reticular accentuation, nodulation may be coarse (and fairly open) or fine (and closely packed), "soft" or "hard" in appearance. Though the nodules are discrete there is the same diffuseness, regularity of size, and uniform symmetrical distribution from apex to base and to the periphery of the lungs in a typical film. The same occasional tendency to localization to the upper halves or middle zones of the lung fields is noted.

With both reticular accentuation and nodulation, the coarser, softer and more open type tends to appear earlier and among younger men than the finer, harder, closely packed variety, and in mines or districts where the free silica content of the dust inhaled is high. After their first appearance, nodules tend to become larger and more plentiful in succeeding years, particularly if the subject continues to work in the industry.

Coarse types are on the whole more common in Western Australia than the finer types of fibrosis as seen in the radiograph. The local distribution suggests that the finer and more closely packed types of nodulation, resembling the "reticulation" of coal miners, are the result of long exposure to mixed dust of comparatively low silica content, but perhaps containing silicates or other so-called "inert" dusts.

Rate of Development.

The onset of pneumonokoniosis is very gradual. The observed gradation in the average machine miner in Western Australia, inhaling dust of free silica content of from 20% to 30%, is, with present standards of ventilation, wet drilling, watering down and restrictions of shot firing, from normal to definite reticular accentuation in eight to ten years after entering the industry, and to classical nodular silicosis after sixteen to twenty years' mine work. After twenty years a very considerable proportion (about 30%) of the men continuously in employment as machine miners have developed nodular silicosis. Some men, apparently starting with exceptionally efficient nasal and bronchial filters and lymphatic systems, or being exceptionally careful in the use of methods of prevention available to them, are unusually resistant, and do not progress beyond the stage of reticular markings in a lifetime underground.

Increased Fibrosis.

The term "increased fibrosis", as used at the laboratory, embraces all stages of development of industrial fibrosis, as seen in the radiograph, up to the borderline of "primary" or classical nodular silicosis. At the latter stage nodulation dominates the X-ray picture and the reticular pattern appears to have disappeared or faded into the background.

About 250 separate individuals throughout the industry are newly diagnosed as having increased fibrosis each year, and experience shows that from 10% to 15% of these, if they remain on the mines, will eventually develop the nodular dominance of primary silicosis. The remainder do not progress beyond reticular accentuation, and there is evidence that only a very small number of men with reticular accentuation who leave the mining industry at this stage progress to a disabling nodular silicosis. As a rule, the earlier the onset of reticular accentuation, the more likely is a person to develop silicosis and the earlier thereafter will it appear.

Slower onset of reticular accentuation of non-progressive type characterizes all types of non-machine mine work, where the men are working in relatively lower dust concentrations. A percentage of these men do, however, develop classical nodular silicosis after from twenty to thirty years underground.

Tuberculosis.

The incidence and course of tuberculosis appear to be about the same in men who have reticular accentuation as in "normal" persons. About one in every four men with established nodular silicosis will eventually become tuberculous. Even when tuberculosis supervenes on an already fully established silicosis it frequently undergoes periods of arrest as in younger men free from dust disease. Eventually, however, it leads to spreading consolidation and cavitation, not infrequently in multiple foci, often at first unilateral or asymmetrical, and most commonly situated either at one apex or in the lower third of the lung field, spreading out from the hilus.

The appearance of "fluffy nodulation" is generally a sign that frank tuberculous disease will develop within a few years. Likewise, the sudden appearance of nodulation within twelve months or two years, especially if there is absence of the intermediate stage of reticular markings, usually has the same portent. In such cases a localized focus is often visible at one or other apex, which may, however, become apparently arrested for many years. In these cases there are often few or no symptoms and the usual clinical signs of tuberculous disease are absent. Years later the tuberculous process may again break down, though it does not do so invariably.

Symptoms.

So far as pneumonokoniosis is concerned, the only essential symptom is progressively increasing shortness of breath on exertion. Machine miners usually first find difficulty in getting to or from their working place when this entails climbing 50 feet or more of vertical ladders. Such dyspnœa is very considerably increased if tuberculosis supervenes or if bronchial obstruction or emphysema is present.

As the breathing becomes difficult, the patient may experience muscular pains about the thorax and shoulders.

Cough and expectoration are absent unless there is associated bronchitis or other infective complication. When present the cough is of irritating type, severe at night and in the early mornings, and causes loss of sleep, in some cases leading to a considerable degree of nervous upset. Bronchitis may complicate either the reticular or nodular type of pneumonokoniosis and lead to incapacity, which, however, in the majority of cases is not permanent, unless the condition is very firmly established. Six to twelve months out of the industry, especially if the person is able to find employment in the open air, leads to clearance of the bronchitis and of a great deal of the tight feeling of the chest.

Loss of weight and strength, loss of appetite and like symptoms are likewise not a feature of simple uncomplicated dust fibrosis. These are present only when infection, commonly tuberculosis, has supervened.

Physical Signs.

With uncomplicated reticular type fibrosis, physical signs of disease (and evidence of incapacity) are conspicuous by their absence. The condition is detectable by radiological means only. As nodulation develops, along with the development of symptoms, the miner commences to show clinical signs and evidence of incapacity. These are, briefly, increased respiration rate on exertion, symmetrical impairment of percussion note, harshness of the breath sounds with prolongation of the expiratory phase, and a peculiar and almost specific flattening, with block movement during respiration, of the upper part of the anterior chest wall. When emphysema, bronchitis, pleurisy, costo-spinal arthritis, tuberculosis or heart disease is present, the signs of these may modify the above picture. In uncomplicated pneumonokoniosis the vital capacity becomes progressively diminished.

Complications.

In Western Australian gold miners well established and persistent bronchitis and clinical emphysema of hypertrophic type, when present in association with nodular fibrosis, are regarded as part and parcel of pneumonokoniosis, unless there is evidence to the contrary. There are also occasional cases of persistent bronchitis in comparatively young men in which radiologically no more than reticular fibrosis is present. In my experience a transient irritating cough is not uncommon at this stage.

In the radiograph there is a tendency for emphysema to mask fibrosis, and due allowance must be made for this possibility. With serial films this is not difficult.

Such associated conditions will considerably enhance the intrinsic incapacity of nodular or massive fibrosis, and when associated with reticular fibrosis will sometimes lead to incapacity where otherwise none would exist.

Evidence of cardiac embarrassment is rare among working miners, and although probably quite a few silicotic miners eventually die from either respiratory or cardiac failure, it is the experience of the writer that circulatory failure occurs only in the presence of nodular or massive fibrosis or gross emphysema. Even in these cases there is nearly always evidence of some contributory condition, such as arterial hypertension or degenerative disease.

When pneumonia occurs in the presence of pneumonokoniosis there is usually at least partial failure of resolution, resulting in a permanent local increase in scar tissue, and most of the massive shadows occasionally encountered under present-day conditions, other than those of a progressive nature indicative of superadded tuberculosis, are probably the result of this process. Attacks of pleurisy, similarly, usually leave adhesions, and incapacity tends to develop or become aggravated after attacks of these diseases.

In my experience the onset of tuberculosis superimposed on pneumonokoniosis is usually insidious, and only rarely do we see it in sufficiently progressive form at the outset to lead to early aggravation of symptoms and physical signs. Records show that in years past, apparently because of this fact, a number of minimal lesions have been observed to appear, harden up and calcify and become apparently fully arrested without the slightest evidence of symptoms or ill health, the men having continued at work throughout. In some such cases, even in the presence of established silicosis and continued exposure to further dusting, there has been no further breakdown of the tuberculous process during the man's working life.

Industrial Exposure.

In regard to the duration of employment there are, as indicated earlier, notable variations in the average time in which silicosis develops in different districts.

In a recent survey of 200 new cases of nodular silicosis, the average period of employment underground before the onset, for the whole of the Western Australian goldfields, was seventeen years for machine miners, and twenty-two years for other underground occupations. These average times applied fairly consistently throughout the goldfields except in two centres, Mount Magnet and Norseman. At these two places, where the quartz content of the dust inhaled is very high, the average time in machine miners was ten years. The average period on the "Golden Mile", where some 60% of silicosis cases arise, is sixteen to seventeen years for machine miners. Under present day conditions 75% at least of all new cases of silicosis are in men who have been machine miners. The remainder come mainly from other underground occupations, mainly timber men, boggers and truckers, and men employed on ore passes.

Assessment of Incapacity.

The assessment of incapacity is the most difficult phase of work in industrial pulmonary disease, chiefly because, although a great deal of attention has been given to the subject overseas, there are still no set rules available for guidance, nor has anyone yet succeeded in evolving a completely objective quantitative clinical test of pulmonary capacity.

After considerable experience I have found that the X-ray film gives an accurate indication of the degree of pathological change present, in so far as fibrosis is concerned, and can safely be used as a guide to the extent of incapacity due to simple fibrosis. If clinical incapacity is actually greater than one would expect from the X-ray film, this is invariably due to the presence of associated conditions, most commonly bronchitis with or without spasm, emphysema, or tuberculosis, which can themselves be detected and assessed largely by clinical means, or else to non-industrial disease.

To sum up, in my view, assessment of incapacity in pneumonokoniosis requires a summation of incapacities due (*a*) to the pure fibrosis as detected by X-ray examination, and (*b*) to any related clinically detectable condition present. Considered as a pure fibrosis, the intrinsic incapacity from reticular accentuation is nil. Partial incapacity appears with commencing nodulation. With nodular dominance the subject usually becomes unfit for further underground work, though by no means totally disabled. Advanced massive shadows usually, though not always, indicate total incapacity. All men with tuberculosis are regarded as totally incapacitated whilst the disease is progressive.

Onset of incapacity which can be confirmed by positive clinical signs occurs in affected machine mine workers in Western Australia after fifteen to twenty years underground, rarely earlier, frequently later.

The most difficult cases are the comparatively large group in which genuine symptoms are present, together with a non-industrial malady and X-ray evidence of a minor grade of industrial pulmonary fibrosis (reticular accentuation). In these cases the assessment hinges on the question of possible aggravation of the non-industrial condition. The extent of such aggravation is often very much a matter of opinion, but if a genuine possibility of this exists, the worker who, though not ill from dust, has manifestly received some damage to his pulmonary lymphatic system, is usually in practice given the benefit of the doubt. The use of electrocardiography, X-ray examination of antra, Wassermann test and other ancillary investigations is often of great assistance in determining the significance of symptoms and the relation to one another of pneumonokoniosis and other non-industrial conditions. Such investigations are made use of at the Kalgoorlie laboratory according to the indications found in individual cases.

Prevention.

For various reasons a good deal more attention is now being given in the Western Australian mining industry to the prevention of pneumonokoniosis than for some time past. The application of old and tried methods is being intensified, and new methods, including water jets, road consolidation, and the use of aluminium dust prophylaxis, are being or will be tried on an increasing scale.

Statistical material for the assessment of the effectiveness of these methods is provided by the results of the periodic medical examinations of working miners conducted by the laboratory. In this regard a study of the prevalence and incidence of reticular markings in different localities and mines from time to time should give useful information in about half the number of years which would be required for a similar assessment on the basis of silicotic nodulation.

Summary.

1. Some figures prepared from the records of the Commonwealth Health Laboratory, Kalgoorlie, are given which serve to show the prevalence of industrial pulmonary disease on Western Australian goldfields over the past twenty-three years.

2. Since the inception of the laboratory in 1925, 1804 cases of simple silicosis and 839 cases of tuberculosis with or without accompanying pneumonokoniosis have been diagnosed.

3. The condition of reticular accentuation is very widespread amongst underground mine workers and is almost invariably

symptomless. The prevalence of this condition in gold miners has considerable significance in relation to the prevention of silicosis.

4. The significance of industrial history and of radiological and clinical findings in the diagnosis of pneumonokoniosis is discussed in the light of local experience.

5. An endeavour is made to set down the principles which guide the writer in assessment of disability.

Reports of Cases.

A CASE OF STRICTURE OF THE JEJUNUM, WITH INTESTINAL OBSTRUCTION AND PERFORATION.

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THE following case appears to be unusual and worthy of publication.

Clinical Record.

L.H.P., a man, aged sixty-one years, was admitted to the Launceston General Hospital on March 10, 1948, complaining of eighteen hours' abdominal pain and vomiting. He had been awakened at about midnight the previous night by a "cramp-like" pain across the whole of the lower part of his abdomen, which had persisted unchanged ever since. About an hour after the onset he had started to vomit and this had continued. He had not noticed any abdominal distension, but his abdomen felt tight. His bowels had not opened since the onset of symptoms. He had no urinary symptoms. He had suffered from periodic similar attacks for two years. These attacks would last one to three days and then remit spontaneously, but this was the most severe he had had. The attacks had not been very frequent at first, but had occurred four or five times in the last month. Between attacks he felt well and had a good appetite and no digestive disturbances. His only other previous medical history was that he suffered from generalized chronic arthritis, and there was no relevant family history.

On examination he was emaciated and had a marked flush on his face, with sunken, staring eyes. His temperature was 100° F., his pulse rate 92 per minute, his respiration rate 24 per minute. His tongue was dry and coated and his breath offensive. His abdomen was slightly distended and rigid in all areas, slightly more so on the right side. His abdomen was tender in all areas, particularly at points below the right costal margin, in the right iliac fossa, and in the right groin. No mass or viscus was palpable through the rigid abdominal wall. There were no abdominal scars, and no herniæ were found. The area of liver dulness to percussion was within normal limits. *Per rectum*, a vague resistance was palpable in the recto-vesical pouch of peritoneum; otherwise no abnormality was found. He had coarse râles at the base of his right lung and general chronic arthritis; physical examination otherwise revealed no abnormality.

Acute general peritonitis was diagnosed, with probable right basal pneumonia, and immediate laparotomy was decided upon. Opinions on the primary abdominal lesion included acute appendicitis, perforated duodenal ulcer, and strangulated internal hernia.

At operation, the peritoneal cavity was found to contain a large amount of purulent fluid, with a thick fibrinous deposit in many places. The pelvic coils of bowel were plum-coloured and covered with fibrin. In the left iliac fossa moderately distended coils of bowel were seen; these terminated abruptly about eight feet from the duodenal flexure, and gave place to empty, collapsed bowel. At the transition there was a hard, rubbery, fibrous mass involving the bowel wall; immediately proximal to this was a small gangrenous patch, which had perforated. About seven inches of bowel were resected. Unfortunately the patient collapsed and died shortly afterwards.

The mass proved to be a dense fibrous stricture, about half an inch long, encircling the bowel, and thickest opposite the mesenteric attachment. Immediately above the stricture, close to the mesentery, there was a small ulcer, with a necrotic, perforated base, and surrounded by atrophic mucosa.

The microscopic report on the stricture, by Dr. A. H. Tebbutt, of Sydney, was as follows.

> The section shows a diffuse infiltration of the wall of the bowel, mainly by small round cells. There are occasional cells with large hyperchromatic nuclei, and a follicle with epithelioid cells and a giant cell with peripheral nuclei. In the absence of any other lesion I find myself unable to give any definite opinion, except that I do not think it is neoplastic.

Dr. R. Y. Mathew, of the Commonwealth Health Laboratory at Launceston, was unable to demonstrate tubercle bacilli in the section.

Autopsy showed general peritonitis, with numerous peritoneal adhesions, and confirmed the diagnosis of pneumonia, but showed no lesions in other organs.

Comment.

This condition is evidently not congenital, and there is no history of trauma, such as injury or operation. The microscopic appearance does not suggest malignancy. It may possibly be a low-grade lymphosarcoma, but this seems very unlikely in the absence of enlarged spleen or lymphatic glands. It seems more likely to be a granulomatous inflammation, and syphilis, tuberculosis and Crohn's disease appear to be possibilities.

Syphilis is rarely found in the bowel, but has long been recognized. It is mentioned by Wakeley and Hunter (1943) in "Rose and Carless' Manual of Surgery" as causing strictures, which usually occur in the rectum but may be in the jejunum. Tuttle (1932) reports a case of syphilitic stricture of the jejunum, and reviews the literature of the subject. He concludes that syphilitic lesions of the small intestine are fairly common in the early secondary and late tertiary phases, but they usually resolve under treatment and are not recognized. Late lesions, such as gumma or stricture, are rare, and should not be diagnosed without clear evidence, such as the demonstration of spirochætes in the lesion. He quotes Cheyne and Burchard as describing multiple strictures encircling the gut, usually affecting the upper part of the small intestine. He collects 77 reported cases, including King's series of 33, of which 18 were in the jejunum. However, conclusive proof appears to be lacking in many of the described cases and these would now, I think, be classed as Crohn's disease.

Tuberculosis of the bowel is well known and is well described in the standard textbooks of pathology (Boyd, 1940; Muir, 1941). Primary bowel infection appears to be rare, and usually gives rise to *tabes mesenterica* with minimal bowel lesions. Ulcerative and hypertrophic lesions are almost invariably secondary to lung disease. The disease usually commences in the ileo-caecal region and spreads upwards and downwards in the lymphatics, producing the well known hypertrophic lesions and "girdle ulcers", but this is not invariable. Healing of extensive ulceration may produce strictures, which are often multiple and predominantly affect the lower part of the ileum. It was diagnosed quite commonly until Crohn described non-specific regional enteritis in 1932, since when there have been few cases reported. Hartmann (1934) reported 32 cases in a series of 466 small-bowel lesions encountered over five years at the Mayo Clinic, not including peptic ulceration. One of these cases occurred at the duodeno-jejunal flexure, and was associated with caseating regional lymph nodes. Schwabacher (1936) and Taylor (1945) stress the fact that tuberculosis should always be looked for before a diagnosis is made of non-specific regional enteritis. Hawksley and Meulengracht (1936), commenting on the association of pernicious anæmia with intestinal stricture, reported 25 collected cases, in only two of which were there associated tuberculous lesions. Paul (1944) discusses the differential diagnosis of tuberculous from non-

specific regional enteritis. He states that a tuberculous ulcer or stricture is usually circumferential and may be associated with mesenteric tubercles, caseating glands or other scars. Diagnosis may rest on microscopic examination, but he makes no mention of demonstrating tubercle bacilli, and Crohn and other writers stress the microscopic similarity of the two diseases.

Non-specific regional enteritis was described by Crohn and others (1933) as affecting a variable length of the lower part of the ileum, with often more than one area of bowel involved, and occurring mainly in young adults. The disease starts as acute inflammation, and often runs a chronic course, ending in fistula or stricture formation. Stricture formation is usually multiple. Since the first description there has been a steady flow of literature and many cases have been reported. Jackson (1937) was able to collect 219 cases in the United States of America up to 1937. The original description of the disease has not been materially altered, except that cases of the disease at various stages have been reported in other sites than the lower part of the ileum: the jejunum (Harris, Bell and Brunn, 1933; Brown, Barger and Weber, 1939), the colon (Brown, Barger and Weber, 1939), the appendix (Rose, 1946), and even a Meckel's diverticulum (Norrish and Barrington-Ward, 1937). Crohn (1939) agreed that the disease could affect any part of the small bowel, and often the large bowel as well. He also thought it possible that mesenteric lymphadenitis might be the same disease. He reported 73 cases, in three of which the jejunum was involved, and Brown and McDonald (1941), reviewing 178 cases from the Mayo Clinic, found five involving the jejunum. I have collected 69 other case reports, in five of which the jejunum was involved. The ætiology of Crohn's disease is not yet known, though it is generally accepted as a separate disease entity. Various theories as to its causation have been advanced, but none have been accepted. It seems possible that it is the common result of several factors, and it still seems possible that many cases are originally tuberculous, though Crohn has stressed that the two diseases are entirely separate. In this connexion the remarks of Anderson and Munro (1932) are notable; working at Glenlomond Sanatorium in Scotland, they found that many cases of intestinal tuberculosis showed no other tuberculous pathology. Most of these cases would now be considered non-specific, but in Scotland, where bovine tuberculosis is common, there are probably many cases of true primary enteric infection.

The present case under discussion is very similar to two cases reported by Paul (1944) as tuberculous, and also to a case reported by Elliott (1948), which he considers Crohn's disease. The sharply defined stricture and the small segment of bowel involved would suggest original tuberculosis or syphilis of the bowel, but in the absence of further evidence, it must be regarded as non-specific regional enteritis, or Crohn's disease.

Summary.

A case of stricture of the jejunum is described, and the ætiology is discussed. In the absence of conclusive evidence of a tuberculous or syphilitic origin, it is considered a case of non-specific regional enteritis; this is known to affect the jejunum in about one case in 30.

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Reviews.

A TEXT-BOOK OF GYNÆCOLOGY.

IN the preface to the fifth edition of his "Textbook of Gynæcology", Wilfred Shaw states that the work has been drastically revised to bring it into line with modern knowledge.[1] Although this revision has been more in the form of additions to various chapters than of changes, the result is a marked improvement on the last edition.

The anatomy, histology and physiology are, as before, excellently done, and Shaw has now added some of his recent contributions to the anatomy of the genital tract. The section on the physiology of the ductless glands has been brought up to date, and reference is made to the work of Markee, and of Smith and Smith, on menstruation. The important chapter on gynæcological diagnosis has been improved by the addition of paragraphs on the uterine sound, cervical and endometrial biopsy, aspiration of Douglas's pouch, coldoscopy, colposcopic examination of the cervix, hysteroscopy, examination of vaginal smears for estimation of œstrogenic function and the diagnosis of uterine carcinoma, biological tests for pregnancy, hormonic assay methods, and curettage.

Changes and a number of additions appear in the ensuing chapters, and it will be sufficient to indicate some of these. For the first time, passages are included on such subjects as *lymphogranuloma inguinale*, Bowen's disease of the vulva, mycotic infections of the vulva and vagina, basal body temperature readings in sterility, androgen therapy in dysmenorrhœa and menorrhagia, and Meig's syndrome. Some of these additions have perhaps been introduced too briefly and too simply. For instance, in the discussion on androgen therapy, no mention is made of the ill effects associated with improper or too long administration; and the statement that the treatment of mycotic infections of the vagina is "simple, for the condition responds immediately to irrigations with 2 per cent. gentian violet solutions in water", is contrary to general experience that in a considerable proportion of these cases the condition is most difficult to cure. A new classification of ovarian tumours based on ideas of Schiller

[1] "Textbook of Gynæcology", by Wilfred Shaw, M.A., M.D. (Cantab.), F.R.C.S. (England), F.R.C.O.G.; Fifth Edition; 1948. London: J. and A. Churchill, Limited. 8½" × 5½", pp. 672, with illustrations, some of them coloured. Price: 25s.

and Meig strengthens this section of the book, but it is strange to see four separate tumours listed as "arising from the ovum".

A number of the author's statements, criticized in a review of the last edition, remain. For instance, we still regard as incorrect and misleading the statement that if the result of the Aschheim-Zondek test is positive after fifteen days from abortion of a hydatidiform mole, chorionepithelioma should be suspected; and the direction that when hæmatocolpos is complicated by hæmatometra and hæmatosalpinx, removal of the uterus and tubes is essential to prevent fatal peritonitis. Further we think old-fashioned the suggestion that in the normal technique for gas insufflation the patient requires an anæsthetic. On the other hand, we strongly agree with Shaw when he points out the importance of not performing uterosalpingography in the presence of genital infection; and we agree with his omission of an earlier suggestion that the good results from uterosalpingography come from the preceding instrumental dilatation of the cervix. It is good to see stated in this edition that the modern treatment of cervicitis and glandular erosion of the cervical canal consists of diathermy cauterization, and that the operation of trachelorrhaphy is rarely required at the present day.

The book is clearly and well written, and is pleasant to read, and good features are the introductory and explanatory passages to certain chapters, such as those on anatomy, physiology, biology, endocrinology and radiotherapy, which are particularly valuable to students. The illustrations are plentiful, well chosen, and for the most part of a high standard, especially those taken from Halban Seitz, Stoeckel, Peham and Amreich, and Neuweiler. A series of line drawings from Neuweiler showing the different grades of cervical carcinoma are beautiful examples of visual aid.

The least satisfactory parts of the book are those dealing with bacteriology and chemotherapy. In neither subject is the work yet up to date. The anaerobic streptococci, which in Australia are the predominant cause of the common uterine and tubal infections following abortion and childbirth, and which cannot be insignificant in England, are not even mentioned. Chemotherapy is quoted without reference to its important bacterial indications. Sulphapyridine and sulphathiazole are the sulphonamides mentioned, yet most people replaced these with safer and better compounds years ago. In one paragraph on the treatment of gonorrhœa, these two statements appear: "At the present time treatment with Sulphapyridine or Sulphathiazole is the most favoured", and "the results of penicillin are far better than those obtained from chemotherapy". There is no reference to the use of streptomycin in genital tuberculosis, or of anticoagulants in the prevention and treatment of thrombophlebitis. In a modern text-book there should be a place for a coordinated account of the present-day approach to the investigation, diagnosis and management of gynæcological infections.

Although, as indicated, this edition contains opinions and statements with which we are at variance, the general standard of the work is high, and the book presents in clear and attractive form a great deal of valuable clinical material and sound teaching.

PHYSICAL SIGNS IN CLINICAL SURGERY.

THE eleventh edition of "Demostrations of Physical Signs in Clinical Surgery", by Hamilton Bailey, has been achieved despite all the difficulties and shortages in post-war England and enhances more than ever its international status.[1] That the wards are the greatest of all research laboratories is epitomized by this standard book on physical signs. Modifications and additional illustrations, many in colour, are included in this latest edition, and the illustrations alone make the book invaluable to every medical student and to practitioners who do not count themselves infallible. Nevertheless it is surprising to read: "Thanks to the adoption of Fowler's position in treatment, sub-diaphragmatic abscess is now comparatively rare."

No criticism can be levelled at the book if one accepts the author's statement that it is not a treatise on clinical surgery, but a series of demonstrations on physical signs; as such it must be unequalled in the English language.

[1] "Demonstrations of Physical Signs in Clinical Surgery", by Hamilton Bailey, F.R.C.S. (England), F.A.C.S., F.I.C.S., F.R.S.E.: Eleventh Edition; 1949. Bristol: John Wright and Sons, Limited. London: Simpkin Marshall (1941), Limited. 8¼" × 5½", pp. 444, with 657 illustrations, many coloured. Price: 34s.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Materia Medica for Nurses", by Lois Oakes, S.R.N., D.N., and Arnold Bennett, M.P.S.; Third Edition; 1949. Edinburgh: E. and S. Livingstone, Limited. 7" × 4¾", pp. 386. Price: 8s. 6d.

Includes material sufficient to cover the syllabus of the General Nursing Council of Great Britain.

"Sternal Puncture: A Method of Clinical and Cytological Investigation", by A. Piney, M.D., M.R.C.P., and J. L. Hamilton-Paterson, M.D., M.R.C.S., with a foreword by the Right Honourable Lord Horder, M.D., F.R.C.P.; Fourth Edition; 1949. London: William Heinemann (Medical Books), Limited. 8½" × 5½", pp. 112, with many illustrations, some of them coloured. Price: 15s.

A summary of the present position of the subject.

"An Account of the Schools of Surgery: Royal College of Surgeons, Dublin, 1789-1948", by J. D. H. Widdess, M.A. (Dublin), L.R.C.P. and S.I.; with a foreword by William Doolin, F.R.C.S.I.; 1949. Edinburgh: E. and S. Livingstone, Limited. 9¾" × 6½", pp. 124, with 16 illustrations. Price: 17s. 6d.

An expansion of three lectures given to students.

"Sir William Gowers, 1845-1915: A Biographical Appreciation", by MacDonald Critchley; 1949. London: William Heinemann (Medical Books), Limited. 7½" × 5¾", pp. 122, with illustrations. Price: 17s. 6d.

Based on Gowers's own writings and on personal recollections of his junior contemporaries.

"This Man and This Woman: A Guide for Those Contemplating Marriage", by Frederick W. Brink, D.D.; 1949. Kingswood, Surrey: The World's Work (1913), Limited. Melbourne: William Heinemann (Medical Books), Limited. 7¼" × 5¾", pp. 88. Price: 5s.

The author's intention is to present a realistic approach to the establishment of a successful marriage, avoiding extremes of romanticism and materialism.

"Histopathology of Irradiation from External and Internal Sources", edited by William Bloom, M.D.; 1948. New York, Toronto and London: McGraw-Hill Book Company, Incorporated. 8" × 5", pp. 850, with many illustrations.

One of a series of volumes prepared as a record of research done under the Manhattan Project and the Atomic Energy Commission.

"The Basic Neurosis: Oral Regression and Psychic Masochism", by Edmund Bergler, M.D.; 1949. New York: Grune and Stratton, Incorporated. 8½" × 5", pp. 372. Price: $5.00.

The author deals *inter alia* with twenty-seven clinical pictures of "oral regression".

"Recent Advances in Oto-Laryngology", by R. Scott Stevenson, M.D., Ch.B., F.R.C.S. (Edinburgh); Second Edition; 1949. London: J. and A. Churchill, Limited. 8" × 5¼", pp. 408, with 106 illustrations. Price: 24s.

The author's intention is to stimulate the interest of medical practitioners in the problems of diseases of the ear, nose and throat, and to bring to their notice recent advances in diagnosis and treatment.

"How to Become a Doctor: A Complete Guide to the Study of Medicine, Dentistry, Pharmacy, Veterinarian Medicine, Occupational Therapy, Chiropody and Foot Surgery, Optometry, Hospital Administration, Medical Illustration, and the Sciences", by George R. Moon, A.B., M.A.; 1949. Philadelphia and Toronto: The Blakiston Company. 8½" × 5½", pp. 148. Price: $2.00.

The purpose of the book is clear from the title.

"Hematology for Students and Practitioners", by Willis M. Fowler, M.D., with a chapter by Elmer L. Degowin, M.D.; Revised Second Edition; 1949. New York: Paul B. Hoeber, Incorporated. 9¼" × 6", pp. 552, with 104 illustrations. Price: $8.50.

An attempt to present hæmatology as a part of internal medicine—emphasis is laid on the clinical and therapeutic aspects of diseases concerned.

"The Surgical Clinics of North America" (issued every two months). Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. New York Number. 8" × 5½", pp. 338, with 68 illustrations. Price: £5 5s. (paper binding) and £6 6s. (cloth binding) per clinic year.

The greater part of this number comprises a symposium on abdominal surgery—included are newer diagnostic measures, technical procedures, and physiological and biochemical information.

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PATHOLOGY AND ITS HORIZONS.

WHEN Paul of Tarsus wrote to the Corinthians about the indivisibility of the body—"all the members of that one body, being many, are one body"—he wrote something which can be applied to many forms of human activity and to the practice of medicine as much as to any other activity. It is trite to say that the different branches of medical science are essentially one and that those who confine their activities to one of these branches are not fundamentally different from those who follow another branch. But these things must be said. Such specialties as radiology, pathology and even ophthalmology often seem cut off from other branches of study and a reassertion, a "*credo*", of essential unity has a revitalizing power.

Theodore J. Curphey in October, 1948, delivered a presidential address to the American Society of Clinical Pathologists and entitled it "Widening Horizons in Pathology".[1] He points out that for the past decade there have been two antithetic lines of development in the history of medicine—on the one hand there have been major advances in the field of science with the conquest of many forms of disease; on the other hand there has been an increasing lack of confidence, amounting to true hostility, on the part of many who have benefited from medical science and care. Organized medicine, he declares, faces these facts with "wide-eyed concern" and medical practitioners are beginning to scan the medical horizon, seeking for means of emerging from the wilderness. Laboratory workers are also scanning the medical horizon and Curphey in his address deals with vistas by which they can enlarge their usefulness. He holds the view that the role which the pathologist will play in the future will depend, "not so much on his scientific knowledge or his technical contributions in the field of medicine, but rather in that area concerned with human relationships". This, we take it, means that the pathologist will still have to be expert in scientific attainment and technical skill, but that he will have to manifest these qualities as it were *coram publico*, for all who will to behold and if possible understand. Curphey states that the social philosophy of the pathologist has been greatly at fault for the past twenty-five years. He does not wish to outline a pathologist's vade-mecum on "How to Win Friends and Influence People", but since the pathologist enjoys a "pivotal position" in the medical scheme it behoves him "to seek ways and means of rendering such service". This must mean that in America pathologists come more often into direct contact with patients than they do in this country. It would appear that more may be effected by what Curphey calls the initial approach of the pathologist in this matter and that is by an examination of his own relationships to those about him. One of his first and greatest aims should be "to develop a cooperative team composed of his own workers, and then to correlate their efforts with that of the various departments of the institution that he serves". If he succeeds he will make all the members of one body realize that, though many, they are all one body. Curphey has the missionary zeal of an enthusiast. He thinks that the pathologist should exercise great care in his own personal relationships—one of the frequent criticisms levelled at him, especially in larger and better organized institutions, is that he is unwilling to identify himself with the general professional life of the institution. The truth is that all too often the clinician fails to take the pathologist fully into his confidence and is apt to regard him as a "press the button machine" from whom a reply of "yes" or "no" or "positive" or "negative" may be obtained after the presentation of a specimen of body fluid or tissue. Curphey admits that certain pathologists because of their unfamiliarity with, or the difficulties of, clinical practice are inclined to assume an air of "spurious nobility" in contact with their associates. When they adopt a dogmatic attitude it is easy to understand how resentment may develop towards them.

The relationship between pathologist and clinician is of fundamental importance in the practice of clinical medicine and every medical graduate will give lip service to the establishment of a trusting and helpful relationship. Whether the fault for imperfect relationship lies, as Curphey implies, solely with the pathologist does not really matter to our present discussion. What is needed is a willingness on the part of clinician and pathologist to discover the truth. The pathologist must not be regarded as a last court of appeal. As Curphey puts it, an investigative function must not be converted into a judicial office. Another important point is that mistakes should be acknowledged. This need not be done with the kind of cheerful optimism that was shown by an Australian small boy, playing his "piece" on the piano at a recital of junior students, and making a lot of mistakes; when told *sotto voce* by his teacher not to make so many mistakes, he stopped, turned to the audience of proud mammas and said brightly: "Anyone is liable to make a mistake." If a mistake is discovered there should be no resentment, but rather an inquiry as to how, with the evidence available, the error was made. Opinions and not personalities should be weighed, and we do well to remember that the man or woman who has never made a mistake has never done anything worth while.

No discussion of this kind can fail to conclude with some reference to student education and training. Curphey had much to say about it and indeed the teaching of pathology could form the subject of a separate and long discussion. Pathology is more than morbid anatomy. It

[1] *American Journal of Clinical Pathology*, January, 1949.

has its experimental side and this is bound to attract the attention of the keen young student, especially if it is presented to him in its proper setting. Those of the older generation, however, can look back with admiration to the way in which some of their teachers made of morbid anatomy a study full of interest and, paradoxical though it may sound, a living explanation of a previously obscure clinical history.

In commending Curphey's address to the attention of readers, we would return to his opening remarks—to his view that advances in medical science have not been attended by a concomitant increase in happiness and peace of mind, which are what he calls the psychic components of general health. He deplores the tendency of the medical profession to dismiss the apparent ingratitude of the people as due to their lack of understanding of the problem and to blame the situation instead on the general social unrest of the world at large. He thinks that the profession may have lost sight of its social responsibility to let the public know the true state of affairs. If this failure is corrected honestly medicine will inevitably ask itself whether it has become somewhat hidebound and whether it should not enlarge its horizon and by contact with reality increase its own understanding of the world at large. As a matter of fact much of what Curphey has written may be taken as an allegory and applied to medicine and society in general. Society is one body and those who practise medicine are but members of that body.

Current Comment.

ACCIDENTS TO CHILDREN.

ACCIDENTS occurring to children must continue to be the subject of concern until they are virtually eliminated, and it must be admitted that that desirable state of affairs is not in sight at the moment. We know of no comprehensive locally compiled figures on the subject, but the experience of the Metropolitan Life Insurance Company in New York, according to D. B. Armstrong and W. G. Cole,[1] is that accidents today represent the first cause of death among children; although the accident death rate among insured children in the age group one to fourteen years showed a 29% reduction during a recent fifteen-year period, this decline was not consistent and compares unfavourably with the reduction in child deaths due to disease. These facts emphasize, Armstrong and Cole point out, the responsibility of medical and public health agencies to study, develop and apply effective measures for the control of mortality and morbidity resulting from accidents, especially among children. It must not be felt that the problem cannot be solved. Information on its many aspects is available and much thought has been expended by those who really care about the problem on a practical approach to its solution. Armstrong and Cole set out detailed suggestions for organizing a community in the matter of child safety, which they rightly describe as a community challenge. The purpose of the suggested programme is, firstly, to encourage parents, other adults and older children responsible for the health and happiness of younger children to recognize the accident hazards confronting young children, to provide and maintain safe conditions for the child in the home and at play, and to help the child, through example and guidance, to develop safe practices; secondly, to encourage public health, medical, and other interested agencies to give added emphasis to child safety in their own programmes. No useful purpose would be served by reproducing details of the suggested programme; many of the ideas would need to be modified in accordance with local conditions and those actively interested would be advised to study the details for themselves. It is sufficient to draw attention to this study and to emphasize that the problem needs a broad approach; splendid as has been the work of bodies concerned with certain aspects of the problem, such as road safety, an appreciation of the over-all problem and a sense of community responsibility are needed.

One serious and distressing phase of the problem which has not received the attention it deserves is that of burns and scalds. Leonard Colebrook and Vera Colebrook[1] state that in the early part of this century deaths from burns and scalds (presumably in Britain) totalled over 2000 per year, 70% being among children; the figure has steadily declined, but is still about 1000 per year in England and Wales. Moreover, it cannot be assumed that the accident rate has fallen proportionately as modern treatment has reduced the relative death rate. Accidental burns and scalds still constitute a serious problem and a reproach. In an attempt to understand the problem the Colebrooks have made an analysis of the first 1000 patients admitted to the Medical Research Council burns unit at the Birmingham Accident Hospital in the period from 1945 to August, 1948. They state that 70% of the patients received the injury at home and only 30% at work; 70% of the home accidents happened to children, those under the age of five years being most affected. About half of the home accidents were judged to have been preventible, that is, they would not have happened if there had been reasonable care and foresight on the part of the adult concerned. The expenditure on hospital services for these 1000 patients was very heavy; in spite of exceptionally favourable circumstances for their treatment, they occupied beds for no less than 34,582 days. If the home accidents judged to have been preventible had been prevented, this occupancy of beds would have been reduced by 13,304 days. It is estimated that in England and Wales alone each year injuries of this kind must require at least 1,000,000 hospital bed-days. In their examination of the causal factors of these burns in relation to preventive measures, the Colebrooks reveal a fact of considerable interest. They discuss in some detail the report issued after a Home Office inquiry into the subject in 1908-1910, and the point emerges that the association in the home of a naked source of heat and highly inflammable garments worn by women and female children and the younger boys is still the ultimate cause of the most serious accidents, as it was when the Home Office inquiry was made. An important part of the Home Office committee's report related to the high degree of inflammability of flannelette and to methods of fireproofing this material. An effective method of fireproofing by impregnation with stannic oxide had recently been introduced, but for reasons which now appear quite inadequate, the method was not recommended to the Government. If it had been recommended and its introduction effected in the right way, it is likely, the Colebrooks stoutly assert and apparently with justification, that the lives of some thousands of children would have been saved in the intervening forty years, and many more spared the terrible results of non-fatal injuries. The committee piously stated in their report: "Legislation of the kind suggested [that is, for fireproofing of materials] is the less justifiable in view of the fact that matters may, in course of time, be expected to right themselves." "Today, forty years later", the Colebrooks comment, "matters have not righted themselves. . . . Actually progress has been backward. Forty years ago a mother could at least buy an uninflammable cotton garment if she was prepared to pay a little more for it; today she cannot." That is apparently the position in Britain and there is nothing to indicate that it is not the position in Australia. Although there are no reliable data on which they can base an opinion, the Colebrooks' first-hand experience of the problem leads them to the conclusion that no less than 100 children in England and Wales each year owe their deaths primarily to the fact that the garment they were wearing could easily catch

[1] *American Journal of Public Health and The Nation's Health*, May, 1949.

[1] *The Lancet*, July 30, 1949.

alight by touching a fire. That means at least 4000 children burnt to death in the forty years and a great many more, perhaps 20,000, suffering the effects of burns short of actual death. Tests have shown that not only flannelette, but many other popular materials, including those of wool and cotton mixture, are highly inflammable. They can, it would appear, be rendered uninflammable now, as they could have been when the Home Office inquiry was held. "This possibility should be reconsidered without delay." This aspect of the subject is very prominent in the Colebrooks' paper; it obviously rankles, presumably because they have seen at first hand the suffering and waste that could have been avoided. However, they do discuss other important preventive measures, which must not be overlooked. These include improvement in the design of guards for open fires and proper provision for fixing them in all new houses being built by local authorities; regulations requiring that all electric and gas fires offered for sale must conform to certain definite safety standards, which would include adequate guarding; and nation-wide propaganda maintained for several years by radio, by the Press, and by other means. No doubt the approach to the problem differs in some ways in Australia; the problem itself is not identical and available information does not enable us to compare its magnitude with that in Britain as put forward by the Colebrooks. It is a subject that warrants consideration by someone in a position to gather and correlate the facts. We should certainly not be so rash as to suggest that mortality and morbidity from burns and scalds among children in Australia are insignificant or that they could not be substantially reduced.

THE MANAGEMENT OF SMALL BOWEL OBSTRUCTION.

A VARIETY of factors have combined in recent years to improve the management of small bowel obstruction. The application of physiological principles in the adjustment of fluid and electrolyte balance, chemotherapy and antibiotics, better anæsthesia and improved surgical techniques have played their part. By no means of least importance have been continuous suction drainage by means of a duodenal tube, a procedure particularly developed and fostered by Wangensteen, and intestinal intubation with the double-lumened tube devised by Miller and Abbott. The history of these two types of intubation is described in papers read at the Annual Session of the American Medical Association last year by J. R. Paine and T. G. Miller respectively.[1] Paine claims that the chief historical significance of Wangensteen's contribution lies in the fact that it marks the beginning of a downward trend in the mortality rate of patients with intestinal obstruction—"the first such trend since the advent of abdominal surgery". He does not claim that the suction treatment alone is responsible for this decline. Full credit is given to other advances, and the importance of operative treatment is recognized and, indeed, emphasized. It will, however, be agreed that suction treatment has greatly aided surgery and has proved of particular value when obstruction is not of mechanical type. Miller, in a rather charming paper, gives a full account of the work of earlier investigators on which the development of the Miller-Abbott tube was based and pays generous tribute to the "energy, perseverance and ingenuity" of W. O. Abbott in perfecting the procedure on a practical basis. The place of the Miller-Abbott tube in both diagnosis and treatment is now too well known to require further discussion. In a comment on it in these columns on May 1, 1948, reference was made to the paramount importance of operative treatment in the management of small bowel obstruction, an importance fully recognized by those responsible for the introduction and popularizing of the Miller-Abbott tube, and one which it seems requires continued emphasis. F. A. Coller and R. W. Buxton,[2] in presenting the results of a survey of 198 patients with acute obstruction of the small bowel, point out that the mortality rate for patients with mechanical obstruction of the small bowel is still high (it was 38·3% in their series), a fact which they attribute largely to delay in diagnosis. Early diagnosis can be made only by the giving of more attention to the history, physical examination and X-ray examination of patients with abdominal pain, nausea and vomiting; and if early diagnosis is made, operation should be performed. If the patient is examined later, when distension, dehydration and infection have supervened as a late stage of the disease, these complications must be combated in preparation for operation. Now all the modern advances have a chance to display their value, and great value it is; but, as Coller and Buxton point out, perhaps obviously, but none the less urgently, cure can be effected only by operative correction of the mechanical obstructing factor. Advances in our management of the complications of obstruction have tended to obscure this fact, and a reemphasis on the fundamentals of the clinical aspects of early recognition and prompt operative therapy will, they believe, "importantly and favorably affect the treatment of obstruction of the small bowel".

A similar emphasis on the importance of early diagnosis and correct assessment of the patient's condition is made in a concise and helpful discussion of the subject by W. Ross McCarty.[1] His system of classification is interesting and to the point: first into external and internal obstruction, and then, in each respective group, into cases in which the diagnosis has been established early and physiological changes are minimal, and those in which the diagnosis has been delayed and the distension and electrolyte imbalance are pronounced. The early diagnosis of external incarcerated herniæ can be made readily, but internal obstruction involving the small intestine presents more difficulty. As McCarty states, the possibility of intestinal obstruction must be borne in mind in all cases of ill-defined abdominal pain, whether intermittent or constant, especially when previous abdominal surgery has been performed. In the management of the condition, McCarty is a keen advocate of intestinal intubation, not only for treatment, but also for diagnostic purposes, and he considers it logical that a tube should be passed on all patients, whether the stage of obstruction is early or late. In some cases surgical operation may be avoided by this means, but he assents to the general principle that, in the absence of distension and electrolytic imbalance, patients with early obstruction should be operated upon soon. Intubation, if used, requires adequate supervision and supportive treatment. X-ray observation of the progress of the tube is most important, as has been shown by John Devine in this journal. For the late stages of obstruction, McCarty considers, immediate surgical intervention is not indicated, as the resultant mortality is prohibitive. A ten-year survey of operations for intestinal obstruction at the Third Surgical (New York University) Division of Bellevue Hospital, New York, showed a disproportionately high mortality rate among patients admitted seventy-two hours or more after the onset of symptoms of obstruction; as a result hospital policy was changed so that all such patients are relieved of distension and fluid and electrolytes are brought into balance before operation is undertaken, and in the subsequent series, admittedly small, a striking reduction in mortality has been recorded. To this experience McCarty adds the great technical difficulties of handling a mass of distended bowel and the successful management by the intubation method of a large number of acute small bowel obstructions in soldiers with severe abdominal wounds, when further surgery at the time would have resulted in an increased mortality rate; the three factors combine to make him urge the importance of restoration of electrolyte balance and relief of distension before surgical interference in late cases. That surgical intervention will be necessary in the majority of cases he does not deny, and the preoperative treatment must certainly be considered and thorough. His ideas, though not perhaps new to the experienced surgeon, should interest all who have to deal with this not uncommon surgical emergency.

[1] *The Journal of the American Medical Association*, May 14, 1949.
[2] *Ibidem.*

[1] *The Surgical Clinics of North America*, New York Number, April, 1949.

Abstracts from Medical Literature.

DERMATOLOGY.

Tocopherols (Vitamin E).

J. F. BURGESS AND J. E. PRITCHARD (*Archives of Dermatology and Syphilology*, June, 1948) studied a series of cases of *lupus erythematosus* with a view to determining the value of mixed tocopherol therapy in this disease. They describe eleven cases in detail in which frequent clinical and histological studies were made during tocopherol therapy. They state that natural tocopherols have a definite curative value in *lupus erythematosus*. This effect is directed, at least in part, towards regeneration of the collagenous structure. The authors also noticed the effect of mixed tocopherol therapy on photosensitivity, a common observation in cases of *lupus erythematosus*. In four cases in their series, there was a history of photosensitivity, which was entirely lost after tocopherol therapy. In all cases except in one the patients were treated by administration of tocopherols by mouth and parenterally. The authors claim that superficial *lupus erythematosus*, no matter how long-standing, responds with moderate dosage, 100 to 300 milligrammes daily. In severe cases larger doses should be given, up to 600 milligrammes daily, preferably between meals and with an empty stomach. Also the fat content of the diet should be reduced. Treatment should be maintained after clinical cure with a maintenance dose equivalent to 50 milligrammes of α tocopherol daily. At the same time other aspects of investigation, such as the factor of infection, should not be ignored.

Ammoniacal Silver Nitrate for Fungous Infections of the Nails.

W. J. NICKERSON AND S. J. WHITE (*Archives of Dermatology and Syphilology*, June, 1948) state that fungous infections of the nails are more frequent than has been suspected in the past. The thickness of the nail tissue and its comparative impermeability cause infections of the nails to be among the most resistant to treatment of the superficial mycoses. The organisms responsible may be *Candida albicans* or one of the species of dermatophytes, such as *Tricophyton rubrum* or *Epidermophyton floccosum*. The invading fungus penetrates the nail along the free edge and grows towards the matrix, gradually infecting the whole nail. There is usually no noticeable inflammatory reaction and no paronychial involvement in nail infections caused by dermatophytes. In infection with *Candida* organisms there may be paronychia with erythema, swelling and pus exuding along the lateral margins of the nail bed and even under the lunula. Diagnosis of these infections is a laboratory procedure. Results of treatment of onychomycosis have been disappointing. One of the authors thought that the problem might be solved by using a substance that was fungicidal and also could penetrate keratin. Such a substance was found in ammoniacal silver nitrate. Preliminary experiments were carried out with ammoniacal silver nitrate on laboratory animals. In a clinical trial all proved fungous infections of the nails were treated. The affected areas were painted once weekly with the ammoniacal silver nitrate solution and allowed to dry. The authors state that the treated nail turns intensely black, and progress can be readily followed. The new nail grows in free of infection, and the line of demarcation between the treated nail and new growth is clearly drawn by the staining of the nail. Treatment was carried out for various periods of time. A patient was judged cured by the appearance of the nails, by failure to demonstrate fungous elements in direct examination of scrapings and by negative results from culture. For treatment the ammoniacal silver nitrate must be spread over the nail as a film. The authors state that of sixteen patients treated nine have been cured and seven are improved.

Chronic Symmetrical Impetigo.

S. CRAWFORD (*Archives of Dermatology and Syphilology*, January, 1949) states that chronic symmetrical impetigo was first described by Adamson in 1908 as "a form of chronic superficial dermatitis in circumscribed patches with symmetrical distribution occurring in children". It is seen most frequently on children and young adults, but may occur at any age. It may continue for months or years, is very refractory to treatment and may recur after having disappeared for weeks or months. It is an infection by the hæmolytic *Staphylococcus aureus* of the skin of the feet, trunk and extremities. The patients usually have mild or pronounced xeroderma. A bacterial allergy mechanism combined with dryness of the skin brings about a decided chronicity of the eruption, which consists of superficial, circumscribed, variously sized, rounded or oval, non-exudative, adherently crusted patches, pinkish, yellow-red and rosy red in colour. There is no single method of treatment that is satisfactory in all cases of chronic symmetrical impetigo. Wet dressings of 3% boric acid solution or 1:5000 potassium permanganate used periodically are useful. Topically applied antibacterial ointments are of value. Antibiotic ointments are useful, but occasionally disappointing. Staphylococcus toxoid injections have been disappointing. Sparsely scattered lesions often respond to 1% to 5% aqueous solution of silver nitrate, 1% aqueous solution of methylrosaline, 1% solution of coal tar in acetone, or 0·1% aqueous solution of potassium permanganate. The most useful are 1% strengths of ammoniated mercury, yellow oxide of mercury, chrysarobin or salicylic acid in equal parts of wool fat and petrolatum. Irritation of the lesions is to be avoided.

Keratosis Blennorrhagica and Streptomycin.

R. G. THOMPSON, C. B. WHITE AND H. HARLEY (*Archives of Dermatology and Syphilology*, March, 1949) report that a soldier with *keratoderma blennorrhagica*, which had proved resistant to massive doses of sulphonamide drugs and penicillin, was treated with intramuscular injections of streptomycin. Sulphonamide therapy was considered inadequate in this case when gonococci persisted in the urethral discharge and there was no diminution in the cutaneous lesions after the administration of more than 222 grammes of the drug. No significant improvement was noted after the giving of more than 12,500,000 units of penicillin. Forty-eight hours after the administration of streptomycin in doses of 0·4 gramme given intramuscularly, there was complete cessation of urethral discharge. After sixteen days of therapy, during which a total of 40 grammes of streptomycin was given, there was complete disappearance of the cutaneous lesions on the legs and unquestionable improvement on the palms and soles. Slight improvement was evident in the ungual lesions.

Streptomycin as a Cause of Dermatitis Venenata.

S. A. M. JOHNSON AND HELEN P. DAVIS (*Archives of Dermatology and Syphilology*, February, 1949) state that streptomycin may cause toxic erythema, urticaria, pruritus, exfoliative dermatitis and hæmorrhagic eruptions. They report a case in which streptomycin was found to be the cause of acute *dermatitis venenata* of the hands and the eyes of a nurse, who had administered solutions of the drug for six months before any real symptoms of cutaneous sensitivity became apparent. Individual sensitivity was demonstrated by strongly positive reactions to patch tests on the patient contrasted with negative results of patch tests on eleven nurses who also handled the antibiotic under similar circumstances. Removal of the nurse from the floor where she was making contact with streptomycin brought immediate relief.

Psoriasis and Adrenocortical Function.

F. REISS (*Archives of Dermatology and Syphilology*, January, 1949) presents a preliminary report on a possible steroid hormonal ætiological relationship to psoriasis. He states that hypofunction of the adrenal glands has been favoured by many investigators in this regard. This assumption is based not only on the favourable response to adrenal cortex therapy, but also on the observation that psoriasis is accompanied by vitamin *C* depletion and responds favourably to its administration. Hypercholesteræmia, generally associated with psoriasis, is considered to be due not only to hypothyreoidism and altered hepatic function, but to diminished activity of the adrenal cortex. The author quotes the fact that Block and Rittenberg demonstrated, by using cholesterol tagged with deuterium, that not only bile acids but also progesterone is formed from this steroid. Twenty-five cases of psoriasis were investigated as to a possible adrenocortical disturbance. The author states that the increase in the blood level of cholesterol in psoriasis may be of greater significance than has been hitherto considered. The fact that cholesterol, one of the building stones of cortical steroids, may be present in increased amounts not only in the serum, but also in the adrenals, could be interpreted as indicating a disturbed corticohormonal function. In most cases there is an appreciable vitamin *C* depletion; it is conceivable that this depletion may be related to deficiency of adrenal cortex, which normally contains large amounts of vitamin *C*, which is essential for its normal function. A possible further indication of disturbed corticosteroid hormonal function is the diminished excretion of 17-ketosteroids. The diminished excretion of 17-ketosteroids in psoriasis may be due largely to diminished adrenal cortical function.

The disturbance in keratin formation in psoriasis may well be associated with a disturbed corticoadrenal function. Adrenal cortical tumours with increased hormonal production are accompanied by hirsutism. As the chemical content of hair and epidermal keratin is similar, the possibility exists that hirsutism on the one hand and parakeratosis on the other are expressions of hyperfunction and hypofunction of adrenal steroid hormones respectively. The author states that the increased sedimentation rate which he found in these cases indicates an infectious or toxic cause for the disease; this seems a likely possibility, based not only on the observation that removal of septic foci is curative in certain cases, but also on the author's recent observation that psoriasis developed in five cases after vaccination against smallpox.

UROLOGY.

Retropubic Prostatectomy.

T. D. MOORE (*The Journal of Urology*, January, 1949) reports on his experience with 46 consecutive cases of Millin's operation of pre-vesical prostatic adenomectomy. In an endeavour to evaluate this operation in comparison with transvesical adenomectomy and with perurethral resection, a similar number of consecutive cases of treatment by each approach is reviewed. The author considers that the comparison is fair in that all of the patients were of a similar age group and were suffering from similar degrees of obstruction. Examinations, preparation and post-operative care were all along positive and similar lines. There is evidence that the retropubic operation is more easily mastered than perurethral resection, but is somewhat more difficult than the suprapubic (transvesical) enucleation. With the retropubic operation the patients are somewhat more comfortable in the early post-operative period than with the suprapubic operation; healing is prompt, the operation incurs no more risk, and hospital stay is shorter. However, the comparatively frequent occurrence (17% in the author's series) of *osteitis pubis* is a serious drawback. The author thinks that should the prevention of this very distressing complication be possible in future, the new operation may take its place in urological practice, perhaps replacing the transvesical approach for hypertrophies of unusually large size.

Operations on the Solitary Kidney.

C. P. MATHE (*The Urologic and Cutaneous Review*, March, 1949) considers the present-day status of surgical intervention on the solitary kidney and describes various types of conservative operations by which the kidney may be relieved of disease processes and restored more or less to normal function. He states that when both kidneys are present, we have learned that a diseased kidney need not always be removed because its mate is normal. Likewise, in operations on the solitary kidney experience has increased over knowledge, so that nowadays we can conservatively attack many conditions instead of doing nothing at all, since nephrectomy is out of the question. The solitary kidney tolerates operation very well, the recuperative power of the single organ is remarkable and the laws governing compensatory hypertrophy favour prompt healing and rapid restoration of function. The patient can live healthily with a surprisingly small amount of renal tissue. Partial resection of the kidney or semi-nephrostomy is applicable to the removal of quite a variety of diseased conditions affecting one portion of the solitary organ. Localization of the lesion, favourable anatomical conditions assuring good blood supply to the untouched portion, and thorough laboratory and clinical studies enable the urologist to carry out partial resection with a greater measure of safety and gratifying results. Usually the affected portion of the kidney is dilated as a result of localized obstruction, sometimes of a reduplicated ureter. Sometimes one portion of a kidney is very unhealthy from calculous diseases. The author and others whom he mentions have successfully resected the tuberculous half of a solitary "double" kidney. Solitary cysts, and even neoplasms in the solitary kidney, have been attacked by partial resection. Nephrolithotomy, pyelolithotomy, nephropexy, nephrostomy, renal denervation, and renal plastic procedures for hydronephrosis have all been applied successfully to the solitary organ.

Urological Problems of Spinal Cord Injury.

D. J. ABRAMSON (*The Journal of Urology*, September, 1948) considers that the prognosis in cases of spinal injury has improved vastly in the period of the second World War compared with that of the first. He states that a secondary effect of spinal cord injury is the involvement of the urinary tract, with development of a neurogenic bladder. Urinary tract involvement is of overwhelming importance, since most of those who succumb do so because of ascending infection and renal failure. After the injury the stage of retention occurs; this may last only one day or as long as nearly two months, after which reflex micturition occurs. With spinal shock the external sphincter is thrown out of function and the detrusor does not contract. With progressive distension of the bladder and rise in intravesical pressure, a small amount of urine may be forced past the resisting sphincter. The three stages usually described are complete retention, retention with overflow and, finally, reflex micturition. With lesions above the first lumbar segment this reflex micturition develops in about twelve weeks. With *cauda equina* lesions the average time taken is twenty-six weeks. Injuries above the first lumbar segment lead to the eventual production of the so-called automatic bladder. With these supralumbar lesions the bladder reflex is intact, though there is no inhibition from higher centres. A stretch reflex stimulates the bladder to contract, and the higher the lesion the more efficient is the automatic action. There is a tendency to spasm of the external sphincter, more commonly with the lower thoracic lesions, and this may impede micturition. With lesions below the first lumbar segment the sensory and motor pathways are interrupted, with resultant development of the autonomous bladder. Contractions of the organ now depend on local nerve plexuses in the bladder wall. The contractions are poor, but the urinary stream may be fairly good as a result of straining. In the treatment of the recently paralysed bladder, there is little doubt but that the most practical method is high cystostomy. Later, the return of bladder tone can easily be determined by filling the organ slowly and noting whether or not spontaneous emptying occurs. Cystometric measurements are unnecessary. The best method of closing the cystostomy when emptying is established is by surgical excision of the fistula and closure by sutures. If there is contraction or spasm of the vesical wall, or other organic form of obstruction, endoscopic resection is called for. In the general care of paraplegics, the formation of urinary calculi should be guarded against by measures which are well known by now.

Non-Obstructive Renal Insufficiency.

H. E. LEITER, I. G. KROOP AND A. HYMAN (*The Journal of Urology*, February, 1949) discuss modern ideas on the management of anuria and pre-anuric states when the cause is in the kidney itself or on the circulatory side. The kidneys are a major mechanism for control of water, electrolytes, non-electrolytes, hydrogen ion concentration and osmotic pressure. Glomerular filtration and selective activity by the tubules influence the composition of the interstitial and intracellular fluids. Prognosis in acute renal insufficiency is dependent on (i) the reversibility or otherwise of the renal lesion, and (ii) the possibility of maintaining artificially, within physiological limits, the various equilibria of extracellular fluids till recovery takes place. The author states that owing to a wrong outlook in medical circles today, these anuric and oliguric patients arrive at hospital either overloaded with fluids or salts, or in a state of severe acidosis or alkalosis. In the early phase, the use of antibiotics may control infections which can lead to lower nephron nephrosis. Shock is a major cause of tubule damage; it should therefore be prevented and combated early. Isotonic sodium sulphate solution may be useful in cases of intravascular hæmolysis. Paravertebral block with "Novocain" has been advocated to counteract renal vasospasm. In the phase of persistent oliguria or anuria, efforts should be made to maintain fluid and electrolyte balance in the extracellular fluid. About 1000 millilitres of fluid containing glucose are given per day by mouth, if possible, otherwise intravenously. No more should be used, unless it is necessary to make up perspiration or gastro-intestinal loss. Sodium chloride is usually best avoided for fear of water-logging. However, sodium bicarbonate given intravenously is often useful to leave available base for neutralization of acid radicles. The diet should consist almost entirely of fruit juices; the absence of proteins in the diet improves the prognosis. It is possible to remove electrolytes and non-electrolytes by dialysis. The latter may be (i) internal, by gastric or intestinal lavage or by peritoneal lavage, or (ii) external, by passing the blood out of the body through a system of "Cellophane" filters, on the other side of which is a fluid containing the elements of the blood which one does not want to remove; the other elements (for example, blood urea nitrogen) will diffuse through, and thus be eliminated from the blood. The patient is given heparin to prevent the blood from clotting. These methods of dialysis, especially the external method, are called "artificial kidneys".

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Royal North Shore Hospital of Sydney, Crow's Nest, New South Wales, on June 23, 1949. The meeting took the form of a series of clinical demonstrations by the members of the honorary medical and surgical staffs of the hospital.

Dexiocardia.

DR. STUART D. ALLEN presented a female patient, aged seventy years, with dexiocardia. Apart from recent slight shortness of breath on exertion and bronchitis each winter for several years, the patient had enjoyed good health for her age until a week before her admission to hospital, when she developed a cough, breathlessness and pain through the lower part of the right side of the chest on coughing or deep breathing. On examination of the patient, she was found to be a rather distressed elderly woman with cyanosed extremities and a constant distressing cough. Her temperature was 101° F. and her pulse and respiration rates were increased. Chest movement was restricted, with impairment of the percussion note and audible coarse râles at the lung bases. The apex beat was not visible or palpable in the usual situation, but an impulse was felt in the right fifth intercostal space three and three-quarter inches from the mid-line. A diagnosis of bronchopneumonia and dexiocardia was made, and the patient was treated with penicillin and oxygen inhalations. Recovery from the respiratory infection was uneventful, and the patient was then investigated further. The following report was supplied on the X-ray film of the chest: "Dextrocardia present. This appears to be part of a generalised transposition. There is some generalised widening of the aorta." X-ray examination after a barium meal showed the stomach to be transposed, and an electrocardiogram showed the typical changes of dexiocardia with evidence of myocardial degeneration.

Achalasia at Cardio-Œsophageal Junction.

Dr. Allen's second patient, a woman, aged seventy-one years, had been admitted to hospital with a respiratory tract infection. She said that for some ten years she had experienced shortness of breath on effort, but had been well otherwise and had had no symptoms referable to the alimentary system. Five months before her admission to hospital she had commenced to vomit shortly after taking food, but had no nausea or pain in her chest or abdomen. The vomiting persisted for four months and was associated with loss of a considerable amount of weight, but had not recurred during the month immediately prior to her admission to hospital. On examination the patient appeared to have lost weight, but was not wasted. The abdomen was rather prominent, and general discomfort was complained of on palpation, but no masses of viscera were palpable. Apart from crepitations at the base of the left lung, the chest was normal. The apex beat was slightly displaced laterally, and a systolic murmur was audible at the apex. Recovery from the respiratory infection was uneventful. X-ray examination revealed a pronounced degree of dilatation of the œsophagus with achalasia of the cardia. Further investigations were refused, and no treatment was carried out. Dr. Allen said that it was interesting to observe how well the patient's general condition had been maintained over a period of two and a half years, in view of the extreme degree of œsophageal dilatation and the duration of the condition.

Pulmonary Hypogenesis.

Dr. Allen's third patient, a boy, aged six months, had been a full-term, normally developed child. After the first week his mother noticed that he had difficulty in feeding, that his breathing was at times laboured, and that he became slightly blue when he cried. Since then he had gained weight fairly well, but had never reached the normal for his age. Gradually the attacks of shortness of breath accompanying feeding had ceased, and he no longer became cyanosed when he cried. Apart from being below average weight, he was a bright, alert child. On examination at the age of two weeks the child was fairly well nourished and lay quietly with no respiratory distress or cyanosis. His weight was seven pounds ten ounces. Recession of the lower half of the sternum accompanied inspiration. Movement of the left side of the chest was diminished, and there was very little movement of the right side. Vocal fremitus elicited when the child cried was almost absent on the left side of the chest, and the percussion note on that side was hyperresonant. The percussion note on the right side was relatively flat. On auscultation the breath sounds were well heard over the right side of the chest, and were puerile in character; over the whole of the left side of the chest they were greatly diminished. No accompaniments were heard. The pulse had a rate of 120 per minute and was regular. An impulse was visible and palpable to the right side of the sternum in the fifth right intercostal space, about two inches from the mid-line. The heart sounds were normal. There were no significant findings on examination of the alimentary or nervous system, and no developmental abnormalities of the limbs or face. X-ray examination showed "marked displacement of mediastinum to the right". The appearance was "consistent with a ball-valve obstruction of the left main bronchus or a congenital cystic left lung". The child, however, appeared to have less respiratory distress and was taking his food better; it was decided that no further acts of investigation should be carried out and that he should be watched. Since then the physical signs and X-ray appearances had not altered significantly. In view of one severe respiratory infection, it had not been considered advisable to prepare a bronchogram for confirmation of the diagnosis of congenital bronchopulmonary hypogenesis.

Syphilitic Optic Neuritis and Retinitis with Gross Alopecia.

DR. I. A. BRODZIAK presented a female patient, aged fifty-one years, who had been admitted to hospital in January, 1949, with a history of blurring of vision and colour-blindness of two weeks' duration. Except for chronic mild headache the patient had been well until October, 1948, when she was admitted to a district hospital and given a blood transfusion after epistaxis. Two weeks later she had been readmitted to hospital with a rash on her abdomen, generalized hyperæsthesia and rheumatic pains in the limbs and spine, which made her "semi-delirious". Whilst she was in hospital, her hair had rapidly fallen out and she had lost two stone in weight. She had been discharged from hospital early in December, 1948, with her symptoms somewhat relieved. In January, 1949, she had noticed gradual blurring of vision and the onset of colour-blindness. She had a past history of cyanosis, which had been attributed to the habitual use of "A.P.C." tablets. There was no history of venereal infection. On examination, the patient was a dry-skinned, middle-aged woman, with uniform thinning of the hair of the scalp. The nervous system appeared to be normal. Dr. K. B. Armstrong examined the ocular fundi, and reported the presence of mild retinal arteriosclerosis. In the left eye there was pronounced retinitis with concomitant neuritis of the optic disk and a fine vitreous haze. The right fundus was similarly affected, though to a lesser degree. Examination of the visual fields showed no perimetric field defects and no central scotoma; there was a definite failure to distinguish blue from green. Lumbar puncture produced clear fluid under normal pressure. The fluid contained eight lymphocytes per cubic millimetre and was sterile; biochemical examination revealed no abnormality, the result of the test for globulin being negative. The Wassermann reaction was not obtained, and the Lange curve was of a luetic type (1223321000-0). X-ray examination of the skull and chest revealed no abnormality. Blood examination revealed hypochromic anæmia. The urine was normal on microscopic examination. The basal metabolic rate was −9%. The patient was given potassium iodide, five grains three times a day for two weeks, and then ten grains three times a day. A course of bismuth injections twice a week was started, and the patient was referred to her local district hospital to continue the course of antisyphilitic treatment. By the end of April she had made great progress and felt better; her hair had grown on her scalp, and she had recovered her colour vision and her previous visual acuity. She stated that her headaches were "better than they had been for twenty years". The ophthalmic surgeon had recently made the following report: "Discs practically normal (they might go on later to a partial secondary optic atrophy). Some irregularity over each macula and paramacular area, but not very marked."

Hæmochromatosis.

Dr. Brodziak's second patient, a woman, aged sixty-seven years, had been admitted to hospital in January, 1949, complaining of severe shortness of breath on exertion of twelve months' duration, weakness of twelve months' duration and swelling of the abdomen of four weeks' duration. Three months before her admission to hospital she had been passing an abnormally large quantity of urine, but this had subsequently diminished to a subnormal amount. Her

ankles became swollen at the end of each day. On examination of the patient, cyanosis of the mucous membranes was noted, with a rapid respiratory rate, engorgement of the veins of the neck to a level three inches above the *manubrium sterni*, and slight uniform dark pigmentation of the whole body, more noticeable on exposed parts. The heart was clinically enlarged, the blood pressure was moderately increased, and there was pitting œdema of the legs and feet. A smooth, rather soft enlargement of the liver could be felt below the level of the umbilicus, almost to the iliac crest; it was not tender. The spleen was not palpable. The inferior epigastric veins were dilated. Signs of congestion were detected at the bases of the lungs. The blood serum protein content was 6·1 grammes *per centum*, the albumin-globulin ratio being 3·2:2·9. The serum alkaline phosphatase content was 11·2 units; the cephalin cholesterol flocculation test revealed a "one *plus*" degree of flocculation in twenty-four hours, and a "three *plus*" degree in forty-eight hours. The result of the hippuric acid excretion test was normal. The blood urea content was 44 milligrammes *per centum*. The sugar tolerance test produced a curve suggestive of mild diabetes. The electrocardiogram was normal apart from the digitalis effect. X-ray examination of the chest revealed pronounced cardiac enlargement and pulmonary congestion. Examination of a biopsy of the skin revealed the presence of a fine iron-containing pigment in some cells of the *stratum germinativum* and the sweat glands; the deposits were widely scattered. No liver biopsy was carried out. Daily examination of the urine revealed no sugar. Dr. Brodziak said that the patient's condition had improved with the administration of "Digoxin". There was some reduction in the size of the liver, and she was discharged from hospital free of symptoms; but the pigmentation from the iron deposits of hæmochromatosis had remained obvious.

Cerebral Abscess.

DR. F. A. E. LAWES and DR. E. F. LANGLEY presented a male patient, aged twenty-six years, who had been admitted to hospital on March 28, 1949, complaining of anorexia, irritability, lethargy and fever, all of three days' duration. The patient had had a severe head injury in 1939, and had made a good recovery. Apart from occasional frontal headache he had been quite well until March 23, 1949, when his current illness had commenced with a stiff neck and a feeling of being "off colour". On the day of his admission to hospital he became very feverish and vomited. On examination the patient was seen to be a flushed, dispirited, lethargic young man, with a temperature of 102° F. and increased pulse and respiration rates. He had neck rigidity, and the spine sign and Kernig's sign were present. Lumbar puncture produced a clear, colourless fluid under normal pressure, and containing 150 lymphocytes per cubic millimetre and an occasional erythrocyte; the chloride and glucose contents were normal, but the protein content was increased to 70 milligrammes *per centum*. The fluid was sterile. The blood contained 10,500 leucocytes per cubic millimetre, 70% being neutrophile cells. A diagnosis was made of benign chorio-lymphocytic meningitis. His condition soon improved, his temperature approached normal, and the signs of meningitis disappeared. By April 7 he was subjectively well, but his temperature had not quite settled, and he slept all day with his eyes covered. On April 19 he complained of severe pain in the left occipital and right frontal regions. On April 23 a slow personality change was becoming evident, and the diagnosis of a space-occupying lesion was made. By April 27 he was extremely drowsy and apathetic and had photophobia; the left pupil was dilated, and papilloedema was present in both eyes. On April 29, after ventriculography, craniotomy was performed by Dr. E. F. Langley. A needle was inserted into a right frontal lobe abscess, which was evacuated. *Staphylococcus aureus* was grown from the pus. Several aspirations were subsequently performed. The patient gradually became well and was discharged from hospital.

Peripheral Neuritis with Optic Atrophy.

Dr. Lawes then showed a male patient, aged forty-three years, who had been admitted to hospital complaining of numbness and weakness of the legs of two years' duration and of blurring of vision of one year's duration. The patient stated that his mother was in an asylum suffering from chronic alcoholism, and he himself had been a heavy drinker for some twenty years. On his admission to hospital the patient was unable to stand or walk, to read the headlines in the paper, or to recognize small objects. He was poorly nourished and showed signs of mental confusion and disorientation for time and place. Examination revealed wasting of the muscles of both lower limbs with inability to move the right leg at all. The tendon jerks were active on the right side, and the plantar response was extensor. On the left side the ankle jerk was absent and the knee jerk was diminished. There was sensory impairment in both limbs, greater on the left side. Examination of the eyes revealed temporal pallor of both disks. A blood count gave normal findings; the Wassermann reaction was not obtained. Lumbar puncture produced a clear fluid under reduced pressure; it was normal in content and did not yield the Wassermann reaction. X-ray examination of the spine revealed degenerative changes in the disk between the third and fourth cervical vertebræ. The diagnosis was made of peripheral neuritis, the result of alcohol. The presence of the tendon jerk in the right leg and the extensor response suggested involvement of the motor tracts in the spinal cord. Treatment was instituted with vitamin B_1, 50 milligrammes per day, reeducative exercises and occupational therapy. At the time of the meeting the patient was very much better, his colour and general state of nutrition were normal, the psychosis had disappeared and he was learning to walk again.

Uncontrolled Diabetes, Diabetic Gangrene and Vitamin B Deficiency.

Dr. Lawes finally presented a female patient, aged eighty-nine years, with a history of diabetes present for an unspecified period, "sore foot" and "sore tongue and mouth" present for some months, and deficiency of diet due to the fact that she had been "not eating" for some months. On examination the patient was found to have gangrene of the little toe of the right foot, and X-ray examination of the right leg and right foot revealed the presence of advanced Paget's disease and calcification of the blood vessels. The erythrocytes numbered 4,750,000 per cubic millimetre, and the hæmoglobin value was 84%. Treatment was instituted with "Combex", one capsule three times a day, vitamin *C*, 100 milligrammes per day, and a 1700 Calorie anti-ketogenic diet. Insulin was prescribed in dosage determined by four-hourly testing of the urine. Penicillin was given intramuscularly, and a penicillin and "Vaseline" gauze dressing was applied to the toe. The toe was "iced" for fifteen minutes three times a day.

Condition Simulating Cushing's Syndrome.

DR. DOUGLAS STUCKEY presented a married woman, aged twenty-two years, who had been first referred to the gynæcological out-patient department for investigation of amenorrhœa. The Aschheim-Zondek test produced a negative result. She was then referred to the medical out-patient department, where she stated that she had had amenorrhœa for nearly nine months. Her menarche had occurred at the age of twelve years, and the menstrual periods had always been irregular, a small loss taking place over a period of seven days. She was always tired and had no energy. Her weight had increased by three stone in the two years since her marriage, but she had weighed as much as twelve stone earlier in life. A small amount of facial hair had always been present, but the growth had been much heavier during the past six months. There were no other symptoms. She was anxious to have a child. On examination the patient was found to weigh thirteen stone five and three-quarter pounds. She had a florid complexion, facial hirsuties, obesity mostly of the face and trunk, white striæ on the hips and red striæ on the abdomen. The pulse rate was 100 per minute, and the blood pressure was 180 millimetres of mercury (systolic) and 100 millimetres (diastolic). A provisional diagnosis was made of Cushing's syndrome. Further investigation revealed a basal metabolic rate of −8%. The erythrocytes numbered 4,720,000 per cubic millimetre, the hæmoglobin value was 14·8 grammes *per centum* (104%), and the leucocyte count was within normal limits. The radiological appearances of the *sella turcica* and of the spinal column were normal. A glucose tolerance test revealed diminished glucose tolerance. The urinary excretion of 17-ketosteroids amounted to 14·9 milligrammes *per centum* in twenty-four hours, the normal range for an adult woman being 4·0 to 15·0 milligrammes *per centum*. A 1000 Calorie diet was prescribed, with amphetamine sulphate 5·0 milligrammes three times a day. After about four weeks the patient felt much better, weighed twelve stone ten and three-quarter pounds, and had had a menstrual period lasting for ten days with only a small loss. Dr. Stuckey compared the main features of the patient's condition with the classical features of Cushing's syndrome. The characteristic features quoted in the case under discussion were adiposity, amenorrhœa, hypertrichosis, a plethoric appearance of the skin with red or purplish striæ, vascular hypertension, lack of energy and diminished glucose tolerance. Certain features were not evident, but the diagnosis seemed to be fairly clear.

Severed Flexor Tendon Treated by Primary Suture.

DR. H. HUNTER JAMIESON presented a boy, aged five years, who had cut the palmar surface of his right hand on a piece of glass. When examined in the operating theatre within three hours of the injury, the patient was found to have a clean cut at the level of the distal palmar crease involving the flexor tendon, partially severing the sublimis tendon of the third finger, and completely severing both tendons of the fourth finger. The sublimis and profundus tendons were sutured with black silk. The site of union of the severed tendons did not move into the flexor sheath on extension of the finger. The tendons were covered with the remnants of synovial sheath, which were sutured with plain catgut. The wound was dusted with penicillin and sulphonamide powder and closed with black silk sutures. Penicillin was given after operation. The hand was splinted in a position of flexion. The thumb and the first and fifth fingers were not restricted, and active exercise was begun on the following day. The skin sutures were removed on the fifth day, and after three weeks physiotherapy was begun with active exercise for all fingers. At the time of the meeting, seven months after the accident, the patient had full return of function.

Compound Fracture of Tibia and Fibula.

Dr. Jamieson's second patient, a man, aged thirty-eight years, had been knocked down by a motor-car in May, 1948, and had suffered a compound fracture of the right tibia and fibula with protrusion of a tibial fragment, gross soft tissue damage, severe deformity and an area of skin loss approximately two inches by five inches in extent. After minimal *débridement* and reduction of the fracture, the skin was apposed under tension and the limb encased in plaster. Three weeks later, when the plaster cast was removed and reapplied, the skin was found to have sloughed, leaving a defect measuring about four inches by seven inches. In June, 1948, a Kirschner wire was inserted and the limb placed in a Braun's splint, and shortly afterwards the fracture was plated and the limb encased in plaster. Subsequently the skin defect was repaired by a tube pedicle graft from the abdomen via the right wrist. The plate and sequestrated bone were removed in May, 1949, and it was proposed to fill the gap in the bone with chips from the ilium as soon as the skin graft had taken. The X-ray appearances suggested that bony union would not occur, the upper portion of the distal fragment of the tibia appearing to be avascular.

Small Bowel Obstruction due to Gall-Stone.

Dr. Jamieson next presented two female patients, both in their sixties, who had had small bowel obstruction due to biliary calculus.

The first patient had been well until three days before her admission to hospital, when she suddenly experienced severe generalized abdominal pain, at first continuous and then cramp-like. She had persistent vomiting of offensive material, constipation and abdominal distension. On her admission to hospital, she was found to have distension of the abdomen with generalized muscular guarding and tenderness. The intravenous administration of glucose and saline was started, and laparotomy performed thirty-six hours after the patient's admission to hospital revealed a large gall-stone obstructing the lower part of the ileum twelve inches from the ileo-caecal junction. Penicillin, serum and gas-gangrene antiserum were given, and the Miller-Abbott tube and Wangensteen's suction were employed. Her condition was satisfactory for two days after operation; then distension increased and the pulse rate and temperature rose. No flatus was passed. Enterostomy was performed, and the patient's general condition improved slowly during the next few days. She was discharged from hospital thirty days after her admission in very good general condition.

The second patient had had intermitent abdominal pain and vomiting for one week, but no constipation. Attacks of biliary colic had previously been experienced in 1941 and 1945. When admitted to hospital the patient was suffering from continual vomiting and was very dehydrated. The only abnormality found on examination of the abdomen was silence on auscultation. A Ryle's tube was inserted, and continuous gastric suction and intravenous saline therapy were instituted. Immediate investigation revealed a hæmoglobin value of 112%, a blood urea content of 145 milligrammes *per centum* and a blood chloride content of 213 milligrammes *per centum* as chloride and 351 milligrammes *per centum* as sodium chloride. A plain X-ray examination of the abdomen with the patient lying and standing revealed no definite distension of small bowel, and repeated enemata produced good fæcal results. The patient's general condition improved, and two days after her admission to hospital the blood urea content had fallen to 30 milligrammes *per centum*, and the blood chloride content had risen to 312 milligrammes *per centum* as chloride and 514 milligrammes *per centum* as sodium chloride. Two days later the Ryle's tube was removed, but on the following day severe abdominal pain and vomiting recurred, the Ryle's tube was reinserted, and gastric suction and intravenous therapy were recommenced. Four days later a further plain X-ray examination of the abdomen revealed gaseous distension of the small bowel and a large opacity in the right ilio-lumbar region, the appearances being consistent with obstruction due to gall-stones. A gall-stone over 1·5 inches in diameter was removed from the jejunum at operation on the same day. The patient survived a hazardous convalescence complicated by bronchopneumonia, toxic erythema from chemotherapy, and anæmia, which required further blood transfusions.

Carcinoma of Ascending Colon with Pericolic Abscess.

Dr. Jamieson then presented a man, aged forty-five years, who had undergone appendicectomy in January, 1949, for chronic pain in the right iliac fossa. On returning to work three weeks later the patient still felt ill, and after a week had an attack of vomiting and diffuse abdominal pain followed by severe pain in the right iliac fossa, rigors and a raised temperature which settled down slowly. The patient was admitted to the Royal North Shore Hospital in April, 1949, with the provisional diagnosis of appendiceal abscess. For the past month he had noticed a mass in the right iliac fossa and had suffered almost unbearable pain and the loss of three stone in weight. When examined on his admission to hospital he was pyrexial and had a tender mass in the right hypochondrium and lumbar region. Leucocytosis was present, with a normal distribution of leucocytes. Penicillin therapy was instituted. The temperature remained raised and swinging. The patient had attacks of intermittent intestinal obstruction, and the mass increased in size until it pointed and an abscess was opened and drained. The temperature returned to normal, and the general condition improved, but the mass did not decrease in size, and a further abscess had to be drained. Another bout of pyrexia occurred a few days later, a fæcal fistula developed where one of the abscesses had been opened, and phthalylsulphathiazole was prescribed. Further X-ray examination revealed filling defects and irregularity of the ascending colon, the hepatic flexure and the proximal half of the transverse colon. At laparotomy a large, movable mass was found filling the right side of the abdomen. Lateral anastomosis was performed between the lower part of the ileum and the transverse colon. The patient's condition remained unchanged, and laparotomy was again carried out, under cover of phthalylsulphathiazole and streptomycin; the caecum and ascending colon were resected with an obviously neoplastic mass. There was a defect in the lateral wall of the ascending colon 1·5 inches in diameter communicating with the right paracolic gutter. There were enlarged glands throughout the mesentery. The patient's post-operative condition at the time of the meeting was fair.

Subphrenic Abscess.

Dr. Jamieson then presented a man, aged sixty years, who had been admitted to hospital in September, 1948, when a diagnosis was made of subphrenic abscess. The illness had been present for about one month. The patient was treated with penicillin, the condition subsided, and he was discharged from hospital two weeks after his admission. In April, 1949, the patient was readmitted to hospital with a symptomless mass in the right costo-vertebral angle. Signs found on examination of the chest were the same as those found on his previous admission to hospital. This time the abscess was drained and penicillin was again given. Hæmolytic *Staphylococcus aureus* was grown from the pus, and the patient made an uneventful and speedy recovery.

Idiopathic General Peritonitis.

Dr. Jamieson's last patient was a married woman, aged seventy-nine years, who had been admitted to hospital in June, 1948, complaining of vomiting and vague abdominal pain present for two days. She had had one large bowel action twelve hours after the onset of symptoms. Her abdomen was moderately distended and soft, with generalized tenderness greatest in the right iliac fossa, and "release" tenderness. Vaginal examination revealed tenderness in the pouch of Douglas. Two enemata were given, with no return from the first and coloured fluid only from the second. Blood investigations revealed no significant abnormalities. Treatment was instituted with glucose and

saline given intravenously, intestinal suction by means of a Miller-Abbott tube and morphine. A plain X-ray examination revealed considerable dilatation of many coils of ileum and jejunum. Laparotomy performed twenty-four hours after the patient's admission to hospital revealed general peritonitis with purulent exudate in the pelvis, fibrinous exudate throughout the abdomen, and paralytic ileus of small and large bowel. No cause for the peritonitis could be found. Coliform bacilli were grown in culture from a swabbing. After operation the patient had a "swinging" fever which did not respond to chemotherapy, and three weeks after operation a diagnosis of subphrenic abscess was made. Further laparotomy one month after her original admission to hospital revealed that the previous peritoneal inflammation had completely resolved, and no residual collection of pus was found after a thorough search. Gallstones were present, but the gall-bladder was not inflamed. The "swinging" fever became less, but did not completely subside. A fortnight after the second laparotomy purulent vaginal discharge was noticed; it was found that the discharge was coming from the cervix, and that there were slight enlargement and tenderness on movement of the uterus. The discharge cleared up after nearly a week, and with it the fever; the only treatment given had been warm douches. The patient was discharged from hospital two weeks later in her usual state of health.

(To be continued.)

MEDICO-POLITICAL.

THE Medical Secretary of the New South Wales Branch of the British Medical Association writes that he has received as a donation to the Federal Independence Fund the sum of £25 4s. 8d., representing the excess of subscription to a recent reunion dinner of the graduates in medicine of the University of Sydney for the year 1924.

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held in the Anatomy Lecture Theatre, Frome Road, Adelaide, on August 5, 1949.

Plant Growth Substances.

MR. L. A. T. BALLARD discussed some recent views on plant growth substances and their activities. The discussion was confined to hormones of the auxin type and dealt mainly with the naturally occurring hormones of that group. Mr. Ballard said that early work had resulted in the isolation of two carbon compounds, auxin *a* and auxin *b*, containing eighteen carbon atoms, and although indole-3-acetic acid ("IAA") was shortly thereafter isolated from natural sources such as urine, the view was held that "IAA" did not naturally function in the plant as a hormone, or at most played a minor role. Improvements in extraction techniques had established that hormones existed in plants in at least three forms: (i) bound and inactive, (ii) free (diffusible) and (iii) bound and active. With those techniques "IAA" had been obtained from a wide range of plant tissues. In other cases, in which the amounts extracted had been too small to analyse by chemical methods, molecular weight determinations by the diffusion technique had shown the active substance to be "IAA", rather than auxin *a* or *b*. That "IAA" must be regarded as a major, naturally occurring hormone was supported by the discovery of two enzyme systems, one responsible for the conversion of tryptophane to "IAA", the other for the destructive decarboxylation of "IAA". There was a wide range of synthetic compounds which produced effects similar to those brought about by the natural hormones. The picture was simplified in that all those compounds conformed in having (i) a ring with a side chain containing at least one carbon atom in addition to the terminal carboxyl group, (ii) a double bond in the ring adjacent to the side chain, and (iii) a certain spatial relation between the carboxyl group and the double bond. The properties of such compounds were discussed fully in relation to these requirements. Mr. Ballard went on to say that, in view of the great diversity of effects produced by these hormones, search had been made for some "master reaction" controlled by them. Two classes of theory were discussed in that respect. The first recognized the importance of respiration in relation to growth and envisaged auxin as a coenzyme in some energy transfer system. The second regarded the level of protoplasmic organization as of critical importance and interpreted auxin activity in terms of its effect on protoplasmic structure.

The Adrenal Cortex.

MR. I. G. JARRETT in a paper on physiological activities associated with the adrenal cortex presented an outline of the chemistry and biogenesis of the associated steroid hormones.

He discussed the importance of the glucogenic corticoids in relation to gluconeogenesis from protein, and of the desoxycorticosterone group in controlling some aspects of electrolyte balance. He said that the former probably exerted a main effect on the breakdown of tissue protein and the subsequent conversion of the deaminated amino acids to glycogen, while the latter were prominent in altering the threshold of the kidney for the excretion of sodium, chlorine and potassium. Conditions associated with the excretion of the cortical hormone were discussed and the prominent part played by the cholesterol and ascorbic acid present in the cortex was stressed. Mr. Jarrett said that the work of Long at Yale and of Vogt at Edinburgh indicated that the changes in adrenal ascorbic acid and cholesterol might be used as indicators of cortical activity, and that the various conditions that increased the cortical secretion did so in all probability by first activating the adrenotrophic secretion of the anterior part of the pituitary. It had also been shown that the release of adrenaline increased cortical secretion, but whether by a direct effect on the gland or by stimulation of the anterior part of the pituitary was not yet determined.

Correspondence.

AIR TRAVEL AND MEDICAL FITNESS.

SIR: In the leading article in the issue of July 23, 1949, the attention of members was drawn to the question of medical fitness for air travel. It is a good thing for these facts to become more widely known and acted upon by the profession.

There is a tendency, however, for one or two misconceptions to become popular, and it would be well to draw attention to them at this time.

Aeroplanes are designed essentially for carrying normal, healthy people, and it is not the business of operating companies to cater for the sick and injured beyond occurrences in flight. In other words it is not the business of airline companies to provide aerial ambulance services. However, the sick and injured are carried when necessary, provided they are fit to travel by air and provided the patient is accompanied by someone who is capable of looking after the case during the journey. It has to be remembered that air hostesses are, in the main, only trained in first aid and their job is to look after the ordinary passenger. Other obvious essentials are that the case is not one of an infectious disease and will not be objectionable to other passengers.

The paper referred to in the leading article is an excellent one, and few are as capable as Sir Harold Whittingham to deal with such a subject. The remarks in the concluding paragraph concerning flights within Australia are pertinent, and it says much for the conditions of travel here that, apart from accidents, the number of people who have died in flight during the past few years would not run into double figures.

There is a tendency, however, to regard the pressurized cabin as something magical—something which does away with all the hazards of flying. Unfortunately at the present time this is not so. The current designs of pressure cabins do not maintain sea-level atmospheric pressure at all altitudes; what they do is maintain a differential pressure, which is quite a different thing. For example, a pressurized aircraft flying at an actual altitude of 12,000 feet would have a cabin "altitude" of approximately 5-6000 feet—at 17,000 feet the cabin "altitude" would be approximately 8-10,000 feet, and so on.

As the advantage of pressurization is to allow flights at high altitudes, it will be obvious that the present type of pressurized cabin will not avoid the effects of hypoxia and is no safer, from the oxygen therapy point of view, than an

unpressurized aircraft which has an adequate oxygen supply.

No mechanical thing is perfect, and on occasions pressurization equipment will fail. If a pressurized aircraft is flying at a high altitude and the equipment fails, the pilot must do one of two things: (*a*) Descend rapidly to 8-10,000 feet to avoid the effects of hypoxia. If he does this there is a danger of causing barotrauma (traumatic *otitis media*), particularly amongst those with colds or chronic catarrh. (*b*) Descend slowly so as to avoid ear troubles. If this is done there is a risk of ill effects amongst those who suffer hypoxia badly.

One can conclude therefore that, until pressure cabins maintain sea-level atmospheric pressure throughout a journey and are to all intents and purposes infallible, patients will be as safe, if not safer, in an unpressurized aircraft flying as they normally do at 8-10,000 feet, provided there is an adequate oxygen supply.

Yours, etc.,
E. H. ANDERSON,
Medical Director, Australian National Airways Proprietary Limited.

Essendon, W.6,
Victoria,
August 17, 1949.

B.C.G. VACCINE.

SIR: Concerning Dr. Helen B. Wiles's letter published in your journal dated August 13, 1949, the New South Wales Department of Public Health does not control the supplies of B.C.G. vaccine, but acts as an agent for the Commonwealth Serum Laboratories in the distribution of the vaccine. All medical practitioners who are interested can become enrolled as vaccinators after they have attended a demonstration of the procedure and technique carried out on a group. On getting in touch with the Tuberculosis Division of this department, they will be informed when and where the next group is being done. The policy concerning those who are to receive vaccination is laid down by the Commonwealth Department of Health. Those likely to benefit are mentioned in the brochure as being persons of certain age-groups living or working in a potentially highly infected environment, such as trainee nurses and medical students, newly born infants of tuberculous parents and others living in tuberculous households.

It would appear that Dr. Wiles has not distinguished between the Commonwealth and State Departments of Health, as she commences with a reference to the State Department and later makes certain statements from the brochure issued by the Commonwealth Serum Laboratories.

It is not the object of this department to restrain the enrolled vaccinator in any way, but it has undertaken to assist in carrying out the policy of the Commonwealth Health Department in this matter.

Yours, etc.,
MARSHALL ANDREW,
Director of Tuberculosis for New South Wales.

93 Macquarie Street,
Sydney,
August 18, 1949.

THE ROYAL PRINCE ALFRED HOSPITAL CANCER OF THE UTERUS FOLLOW-UP.

SIR: It is necessary to comment on the recent report of Drs. Schlink, Chapman and Chenhall, who are to be congratulated on the excellent quality of this outstanding cancer research. By their thorough study of radical surgery after an application of radium to cancer of the cervix, much valuable data has been produced. It is to be hoped that much fuller reports will be presented in the near future.

The method, as they have applied it, now produces results comparable with those consistently obtained by radiotherapy alone, and in their hands I regard it as a satisfactory alternative mode of treatment. Not many, however, possess the experience and skill of this team.

This work is another step towards the close liaison of the surgeon and radiotherapist, on which, I believe, much of the future improvement in cancer treatment will depend. Study of these and various other sets of figures might suggest at least two ways in which their method could develop. Firstly, the trained radiotherapist, by arranging the preliminary radium to suit the individual case, may well improve the cure rate in operated cases. In the 50% not operated on, the application of radical X-ray therapy to the parametrium would probably save a few more. Of course, radiotherapy requires as much scientific skill and accuracy as surgery in its application.

The statement that radiotherapy does not kill squamous carcinoma affecting lymph glands has been refuted often enough in this journal and one wonders why it is reiterated by these workers. Lymph glands obtained by them far out in the parametrium would consistently receive only a fraction of the known cancericidal dose. Besides, the histopathological interpretation of the appearance of cancer cells after irradiation is not very reliable, as Glucksman's work would indicate. A direct proof of the inaccuracy of the statement cannot be obtained, but the effect on survival rate of well-planned X-ray therapy to the lateral walls of the pelvis seems to be excellent indirect evidence.

I am grateful to these workers for the stimulus they give to our work in Perth, where some of my expressed ideas are being tried out.

Yours, etc.,
ALAN NELSON.

Lister House,
252 St. George's Terrace,
Perth.
August 22, 1949.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 60, of August 18, 1949.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force (Active List): Medical Branch.

The following former officers are appointed to commissions with the rank of Flight Lieutenant, 1st January, 1949: Alan Montgomery Beech (257693), Brian Walle Costello (255909).

The ranks on appointment included in the notifications regarding former Squadron Leader Alva Thomas Pearson (261701) and Dr. William Arthur Newnham (299950) as approved in Executive Council Minute No. 5 of 1949, appearing in *Commonwealth of Australia Gazette*, No. 4, dated 13th January, 1949 (page 83), are amended to read Flight Lieutenant.

Reserve: Medical Branch.

The appointments of the following officers are terminated 31st December, 1948: Temporary Squadron Leader A. M. Beech (257693), Flight Lieutenant B. W. Costello (255909).—(Ex. Min. No. 35—Approved 13th July, 1949.)

Post-Graduate Work.

THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

POST-GRADUATE WEEK.

A POST-GRADUATE week will be held at the Alfred Hospital, Commercial Road, Prahran, and the Women's Hospital, Swanston Street, Carlton, Melbourne, from October 17 to 21, 1949, at the request of the Melbourne Permanent Post-Graduate Committee.

Programme.

Monday, October 17, at the Alfred Hospital.—11.30 a.m.: Dr. E. S. J. King, "Intestinal Diverticulosis", 2.15 to 3.30 p.m.: Dr. J. Eric Clarke, "The Limitations of Radiology in the Diagnosis of Pulmonary Disease"; Dr. C. A. M. Renou, "Experience in the Diagnosis and Treatment of Tumours of the Breast"; Miss Jean Tolhurst and Mr. G. Buckle, "Chronic Ulceration of the Skin by a New Organism"; Dr. Kennedy Burnside, operative demonstration. 4 p.m.: Dr. Ewen Downie, "Diabetes Mellitus: A Retrospect".

8.15 p.m., at the Medical Society Hall, 426 Albert Street, East Melbourne: Professor F. A. E. Crew, Professor of Public Health, University of Edinburgh, "Social Medicine".

Tuesday, October 18, at the Alfred Hospital.—9.15 to 11 a.m.: Dr. D. G. Duffy, "Essential Hypertension"; Dr.

Robert Officer, "Problems in the Management of Carcinoma of the Rectum"; Dr. C. H. Hembrow, "How Frequent are Disk Lesions in Sciatica?"; Dr. John McLean, "Leuchæmia"; Dr. H. A. Phillips, operative demonstration. 11.30 a.m.: Dr. P. Fanti, "The Influence of Drugs on the Blood Coagulation System". 2.15 to 3.30 p.m.: Dr. Morris Davis, "Lesions of the Mitral Valve"—a clinical survey with case demonstration and strip film; Dr. Leonard Ball, "Thyreotoxicosis: When to Operate"; Dr. Charles Sutherland, "The Causes of Asthma"; Dr. Alwynne Rowlands, operative demonstration. 4 p.m.: Dr. H. B. Kay and Dr. C. J. Officer Brown, "Congenital Heart Disease" (patients to be available for examination throughout the day).

Wednesday, October 19, at the Alfred Hospital.—9.15 to 11 a.m.: Dr. H. B. Kay, "Cases of Aortic Valvular Disease"; Dr. Robert S. Lawson, "Problem Cases in Emergency Surgery"; Dr. Leonard B. Cox, "Arterial Disease of the Brain"; Dr. J. M. Buchanan, "Carcinoma of the Uterus: Why the Wertheim?"; Professor A. B. P. Amies, operative demonstration. 11.30 a.m.: Dr. Hugh Trumble, "Surgery in the Treatment of Hypertension".

Thursday, October 20, at the Alfred Hospital.—9.15 to 11 a.m.: Dr. Roderick Andrew, Dr. Ian G. McLean and Dr. Alwynne Rowlands, symposium on "Peptic Ulcer"; Clinical Research Unit, "Some Aspects of Clinical Cardiology"; Dr. Lawrence Stokes, "Sulphonamide Prophylaxis in Rheumatic Fever"; Dr. C. J. Officer Brown, operative demonstration. 11.30 a.m.: Mr. Balcombe Quick, "Hydatid Disease of the Liver" (with colour film). 2.15 to 3.30 p.m.: Dr. J. Erskine Sewell, "Sarcoidosis"; Dr. Russell Howard, "Why Acute Appendicitis is Still Misdiagnosed"; Dr. H. Roy Cash, "Prosthetic Appliances Used for Maintenance of Skin Grafts and for Bell's Palsy"; Mr. W. A. Dott and Mr. Ken McIntyre, "Treatment of the Fractured Jaw"; Mr. Eric Scrivenor, "Immediate Dentures"; Dr. H. C. Colville, operative demonstration. 4 p.m.: Dr. E. V. Keogh, "Immunology: Modern Theory and Practice".

8.15 p.m., at the Medical Society Hall, 426 Albert Street, East Melbourne: Lecture by Professor Alexander Kennedy, Professor of Psychological Medicine, University of Durham.

Friday, October 21, at the Women's Hospital.—9 to 10.30 a.m.: gynæcological operations, (i) Dr. A. M. Hill and Dr. G. Godfrey, (ii) Dr. F. E. True and Dr. C. K. Churches; 11 a.m. to 12.30 p.m.: Dr. D. F. Lawson, demonstration of clinical cases; Acting Professor J. W. Johnstone and Dr. H. F. Bettinger, "Endometrial Biopsy"; Dr. G. B. Bearham, "Primary Adenocarcinoma of the Fallopian Tube"; Dr. A. M. Hill, Dr. H. F. Bettinger and Dr. H. M. Butler, "Post-Abortal Infection by Clostridium Welchii". 2 to 3.15 p.m.: obstetrical operation, Dr. W. M. Lemmon and Dr. L. Townsend, "Cæsarean Section under Local Anæsthesia and Pentothal". 3.45 to 5.10 p.m.: Dr. W. D. Saltau, clinical demonstration; Dr. G. Simpson, "Trilene Analgesia in Obstetrics"; Dr. B. H. Anderson, "The Development of Chorionepithelioma after Hydatidiform Mole"; Dr. W. J. Rawlings and Dr. I. O'Loughlin, "The Importance of Hæmoglobin Estimation in Pregnancy"; Dr. R. McK. Rome, "Recent Advances in the Treatment of Eclampsia".

LECTURES IN SOCIAL MEDICINE.

The Melbourne Permanent Post-Graduate Committee has arranged for Professor F. A. E. Crew, Professor of Public Health, University of Edinburgh, to give four lectures in "Social Medicine" in the Medical Society Hall, 426 Albert Street, East Melbourne, at 8.15 p.m. on Monday, October 17, Friday, October 21, Monday, October 24, and Thursday, October 27, 1949. The fee for this course is £2 2s., or 10s. 6d. per lecture. Resident medical officers may attend at half fees.

SEMINARS AT ROYAL PRINCE ALFRED HOSPITAL.

THE following seminars will be held at Royal Prince Alfred Hospital, Camperdown, New South Wales, on Friday afternoons in October and November, 1949. Each seminar will be held in the A2 lecture theatre between 1.15 p.m. and 2.15 p.m. and will be followed by a pathological demonstration, staff rounds, and, at 4.15 p.m., grand rounds at Professor Lambie's department (Atherden lecture theatre). All members of the medical profession are invited to attend.

October 7: "Secondary Effects of Anæmia"; October 14: "Diagnostic Aids in Pancreatic Disease"; October 21: neuro-

DISEASES NOTIFIABLE IN EACH STATE AND TERRITORY OF AUSTRALIA AND NOTIFICATIONS FOR THE WEEK ENDED AUGUST 13, 1949.

Disease.	New South Wales.	Victoria.[2]	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[2]
Ankylostomiasis	*	..	..	..	4	..	..	..	4
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	1	..	..	2(2)	..	1(1)	..	..	4
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(a)	*	*	*	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	15(14)	..	..	..	..	..	15
Diphtheria	15(8)	..	6(1)	..	4(3)	..	..	..	25
Dysentery(b)	*	..	..	..	1	6(1)	..	..	7
Encephalitis Lethargica	..	..	..	..	..	..	..	..	..
Erysipelas	*	*	*	..	*	*	..	..	..
Filariasis	*	..	..	*	*	*	*	*	..
Helminthiasis	*	..	*	*	*	*	..	..	..
Hydatid	*	*	*	*	..	*	*	..	..
Influenza	*	*	*	..	*	*	..	*	..
Leprosy	..	..	..	..	6	..	..	..	6
Malaria(c)	*	(c)	(c)	(c)	1(c)	(c)	(c)	(c)	1(c)
Measles	*	*	*	34(17)	*	*	..	..	34
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	4(2)	..	1(1)	8(8)	..	..	..	..	13
Psittacosis	*	*	*	..	*	*	*	*	..
Puerperal Fever	..	..	2(2)	..	..	..	..	..	2
Rubella	*	*	*	*	2(2)	*	..	..	2
Scarlet Fever	33(23)	..	13(3)	7(6)	1(1)	4(1)	..	..	58
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	2(1)	*	*	*	*	*	2
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(d)	33(27)	..	11(5)	2	8(6)	7(2)	..	..	61
Typhoid Fever(e)	..	..	2(1)	..	..	..	..	..	2
Typhus (Endemic)(f)	1	..	2	..	1	..	..	..	4
Undulant Fever	..	..	..	..	..	..	..	1	1
Weil's Disease(g)	*	*	*	*	*	*	*	*	..
Whooping Cough	*	*	*	29(6)	*	*	..	..	29
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 36, 1944-1945. Figures in parentheses are those for the metropolitan area.

[2] Figures not available.

[3] Figures incomplete owing to absence of returns from Northern Territory and Victoria.

* Not notifiable.

(a) Includes "Mossman" and "Sarina" fevers. (b) Includes amœbic and bacillary. (c) Statistics inexact with varying practice with regard to relapses in Service cases infected overseas. (d) Includes all forms except in New South Wales and Northern Territory, where only pulmonary tuberculosis is notifiable. (e) Includes enteric fever and paratyphoid fevers. (f) Cases reported include scrub, murine and tick typhus. (g) Includes leptospiroses, Weil's and para-Weil's disease.

logical subject to be determined; October 28: Journal Club (Thoracic Group); November 4: "Overseas Impressions", Dr. J. Halliday (Cardio-Vascular Group); November 11, 18 and 25: subjects to be determined.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following additional qualifications have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales:

Broadbent, John Hayley, Montague Street, Goulburn (M.B., B.S., 1933, Univ. Sydney), D.O., 1949 (Univ. Sydney).

Gunther, William Willis, 17 Bolton Street, Newcastle (M.B., 1938, B.S., 1945, Univ. Sydney), D.D.M., 1949 (Univ. Sydney).

Loewenthal, John Isaacs, 217 Macquarie Street, Sydney (M.B., 1938, Univ. Sydney), M.S., Melbourne, 1946, F.R.C.S. (England), 1946.

Marshall, Samuel Simon, 251 Oxford Street, Bondi Junction (M.B., B.S., 1946, Univ. Sydney), D.D.M., 1949 (Univ. Sydney).

Ward, William Herbert, 17 Bolton Street, Newcastle (M.B., Ch.M., 1920, Univ. Sydney), D.D.M., 1948 (Univ. Sydney).

Booth, Edward Allan, 11 Yallaroi Avenue, Pymble (M.B., B.S., 1940, Univ. Sydney), D.D.R., 1949 (Univ. Sydney).

Loewenthal, Louis Samuel, 217 Macquarie Street, Sydney (M.B., Ch.M., 1926, Univ. Sydney), F.R.A.C.S., 1932.

Obituary.

PETER HALCOMB WALLMAN.

WE regret to announce the death of Dr. Peter Halcomb Wallman, which occurred on August 18, 1949, at Wingham, New South Wales.

CURT ROSENTHAL.

WE regret to announce the death of Dr. Curt Rosenthal, which occurred on August 21, 1949, at Sydney.

Notice.

SCIENTIFIC MEETING ON BLOOD TRANSFUSION.

THE National Council of the Australian Red Cross Society, through its National Blood Transfusion Committee, has arranged a scientific meeting on blood transfusion, which will be held at 8.15 p.m. on Thursday, September 15, 1949, at the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney.

The following papers will be read: "Some Genetical Aspects of the Human Blood Groups", Ruth Sanger, Ph.D. (London); "Pathological Physiology of Purpura", C. R. B. Blackburn, M.D., M.R.C.P. (London), M.R.A.C.P.; "Homologous Serum Hepatitis", R. J. Walsh, M.B., B.S. In addition to the reading of the papers, a demonstration of various aspects of blood transfusion work has been arranged.

All members of the British Medical Association are cordially invited to attend the meeting and to take part in the discussion following the papers.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Taylor, Robert Gregory Darnley, M.B., B.S., 1945 (Univ. Sydney), Whitehall, 28 Great North Road, Five dock.

Medical Appointments.

Dr. George Edward Wardell Streeton has been appointed deputy quarantine officer at Thursday Island, under the *Quarantine Act*, 1908-1947.

Dr. H. J. Ellis has been appointed resident anæsthetist, Royal Adelaide Hospital, Adelaide.

Diary for the Month.

SEPT. 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 7.—Victorian Branch, B.M.A.: Branch Meeting.
SEPT. 7.—Western Australian Branch, B.M.A.: Council Meeting.
SEPT. 9.—Queensland Branch, B.M.A.: Council Meeting.
SEPT. 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 15.—Victorian Branch, B.M.A.: Organization Subcommittee.
SEPT. 19.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135 Macquarie Street, Sydney): Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

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